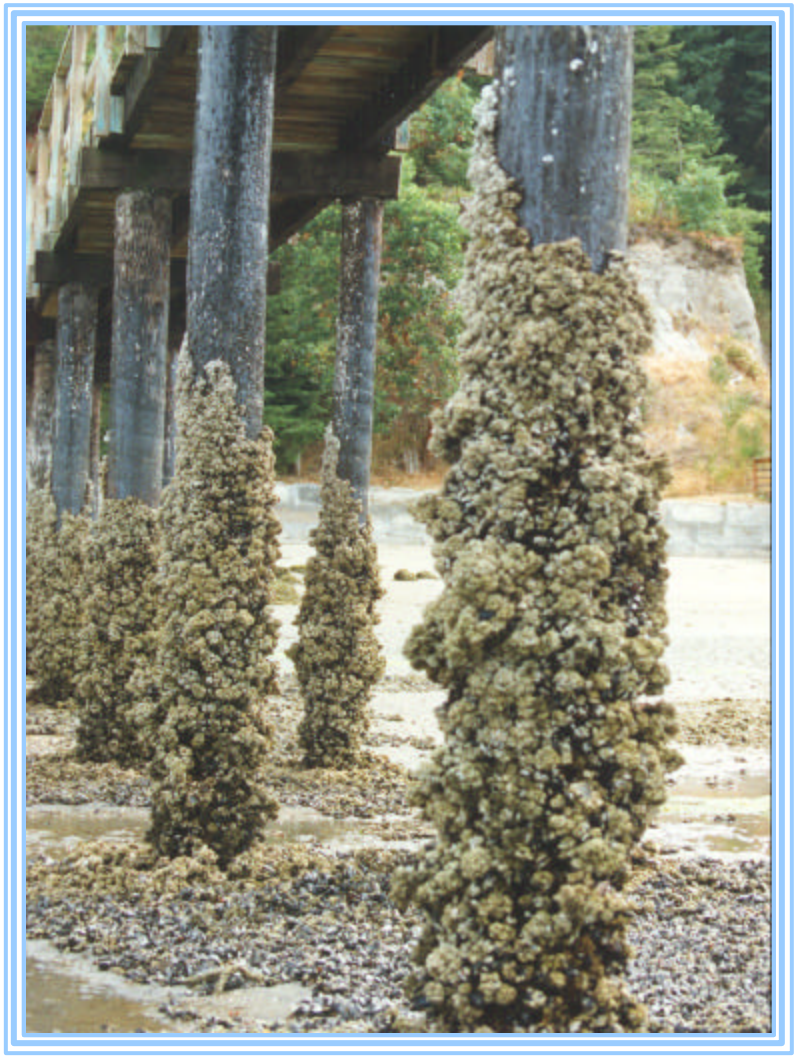
Comments regarding the Environmental Protection Agencies Draft Preliminary Risk Assessment on Creosote



Creosote treated piling at June Beach in Washington State

Creosote Council II

Produced by:

Dr. Kenneth M. Brooks
Aquatic Environmental Sciences
644 Old Eaglemount Road
Port Townsend, Washington 98368

Phone and Fax (360) 732-4464
Email: brooks@olympus.net

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Comments regarding the Environmental Protection Agencies
Draft Preliminary Risk Assessment on Creosote

Kenneth M. Brooks Ph.D.

1.0. Author's qualifications. The author has been studying and modeling the environmental response to a broad spectrum of wood preservatives, including creosote, for over a decade. This work includes development of a field-tested and verified spreadsheet models predicting the environmental response to creosote treated wood projects and completion of five environmental scale risk assessments for the U.S. and Canadian governments (Brooks 1994; Goyette and Brooks 1998; Goyette and Brooks 2001; Brooks 2000b) and industry (Brooks 1999, 2000c). Copies of these documents are appended to these comments.

2.0. Introduction and general comments applicable to the U.S. EPA Draft Preliminary Risk Assessment on Creosote. The creosote draft RED contains several factual errors. However, the most glaring deficiencies result from omission of information – and the focus of this response is to point out those deficiencies and to discuss how the information could be used by EPA to better understand and manage creosote treated wood products. It its present condition, the draft RED does not reflect the available science and it does not provide a rigorous basis for managing creosote treated products.

The compounds of primary concern in creosote are polycyclic aromatic hydrocarbons (PAHs). These compounds are the focus of EPA's RED and they will be the focus of the following comments. There are many sources of these natural compounds and their transport, accumulation in the environment; fate and toxicology have been widely studied. These studies include several that assess actual freshwater and marine invertebrate community response to the presence of real creosote treated wood projects. Brooks (2002) provided a general review of the science applicable to understanding and managing creosote treated wood products in aquatic environments. An additional copy of that document is appended to this response. In some cases that material is copied into this document and in other cases the information has been expanded. My experience, expertise and comments apply only to the following sections of EPA's draft document:

- Residue Chemistry
- Environmental Fate
- Environmental Exposure/Modeling
- Ecological Effects and Environmental Risk Characterization.

3.0. Environmental Fate and Chemistry of Creosote. In some sections, such as that dealing with *Environmental Fate,* EPA's literature review appears to have ended in 1999, resulting in a failure to include significant new literature. For instance, Goyette and Brooks (1998, 2001) conducted a five year study describing the environmental response to six piling dolphins constructed of new and used creosote treated and untreated piling in Sooke Basin, British Columbia. This 568 page document, which formed the basis of the Canadian Department of Fisheries and Ocean's policy regarding the use of creosote treated wood in aquatic environments, was not referenced by EPA. Brooks (2000a) reported the results of a two year study examining the migration of PAH from a rail-line in the Des Plaines River wetland near Chicago. This study examined PAH concentrations adjacent to the operating track and continued with a mesocosm

study to characterize PAH migration to ballast, adjacent wetland soils and ground and storm water from new, used and untreated railway ties. Brooks (2000b) reported the results of detailed environmental risk assessments at creosote treated timber bridges in Indiana along with CCA-C treated bridges in Florida and pentachlorophenol treated bridges in Washington and Oregon.

3.1. Product Chemistry. Goyette and Brooks (1998 and 2001) and Brooks (2000a) provided an opportunity to examine the composition of new creosote oil, the exudates from newly treated products and the environmental fate of parental PAH in railway ballast, wetland soils and parental and alkylated PAH's in marine sediments. The results of these studies consistently demonstrated significant reductions in the proportion of low molecular weight compounds (less than phenanthrene) in comparison with those of intermediate weight. For instance, naphthalene represented 23.8% of the raw oil used to treat the creosote piling produced in accordance *Best Management Practices* (WWPI/CITW, 1996) in Sooke Basin. The proportion naphthalene in the exudates from treated piling was reduced to 10.6% in the BMP piling and it represented 15% of the exudates from eight year old *Weathered* piling in that study. Similar reductions in the proportion of acenaphthylene, acenaphthene and fluorene were observed. Increases in the proportions of phenanthrene and anthracene were observed. Heavier molecular weight compounds remained in relatively constant proportions.

This is important because it clearly demonstrates that the environmental risks associated with creosote treated products cannot be characterized based on the risks associated with the whole oil. These low molecular weight PAH have higher solubility and therefore higher bioavailability with increased acute toxicity in aquatic environments. Their loss during manufacturing strongly suggests that the suite of PAH lost from treated products is less acutely toxic than whole creosote oil. Bioassays and mesocosm studies or studies following creosote oil spills, which examine the biological response to whole creosote oil are akin to assessing the environmental response to plastic by analyzing the various esters that are used to produce the product. EPA should recognize this and include it as part of their assessment of available information.

3.2. Environmental Fate Assessment: Because of their differing physicochemical properties, the composition of the suite of PAH migrating from creosote treated products undergoes further changes, particularly in aquatic environments, following their release from treated products. Goyette and Brooks (1998) and Bestari *et al.* (1998a, 1998b) and Brooks (2000a) all observed a shift in the spectrum of weathering PAH to increasing proportions of intermediate weight compounds (particularly anthracene and fluoranthene) with time and a concomitant decrease in compounds lighter than phenanthrene. Within a relatively short period of time, sedimented PAH derived from creosote were dominated by fluoranthene, with reduced but still significant concentrations of phenanthrene, pyrene, benzo(a)anthracene, chrysene and benzo(b and k) fluoranthenes. Compounds lighter than phenanthrene and heavier than benzo(b or k) fluoranthenes constituted a small proportion of the weathered suite of PAH. The lighter compounds are more soluble and degrade more rapidly than the intermediate weight compounds and the high molecular weight compounds degrade more slowly, but constitute a small proportion of creosote oil at all times. This data is important because there are many sources of PAH and understanding their spectrum in creosote oil as it weathers is useful in assessing the contribution from creosote to the total. For instance, the PAH observed by Wan ((1991) could be derived from the creosote ties, from train cargo (coal in British Columbia where Wan worked) or

from diesel exhaust and lubricants. The relatively higher proportion of phenanthrene when compared with fluoranthene observed by Wan (1991) suggests that uncombusted hydrocarbons (coal) contributed significantly to the PAH mixture and the high proportion of compounds heavier than benz(a)anthracene observed by Wan (1991) suggest contributions from crankcase oil or diesel exhaust (see Figure (1) in Brooks (2000a).

3.3. Mobility. The second sentence of the first paragraph, "Many PAHs adsorb to sediments and may persist for long periods of time." is technically correct but misleading because there is little evidence that this is what typically happens adjacent to real creosote treated wooden structures. The Creorisk Model of Brooks (1994) predicted that the sum of the 16 priority pollutant PAH mixture accumulating in Sooke Basin sediments would peak at ca. 1,000 days post construction and then decline. The five year study observed that the ΣPAH actually peaked sooner (~750 days post construction) and declined more quickly than predicted. While the EPA's statement is technically correct, the RED should be expanded to include empirical evidence describing more likely scenarios.

EPA seems to be confusing creosote migration rates with accumulation and fate in the environment. My own work supports EPA's general conclusion that creosote derived PAH's do not migrate far from treated structures. For instance, in the Sooke Basin Study, statistically significant increases in sediment PAH were restricted to some distance between 7.5 and 10 meters downcurrent from the six piling dolphins and Brooks (2000a) found that PAH migrating from new and used railway ties generally stayed within the ballast. Only very small and biologically insignificant PAH concentrations were observed in mesocosm sediments during this study. I am unaware of any research supporting EPA's assertion that, "Higher amounts of PAHs leached into such soil types (muddy sediments)." Rather, I suspect that higher PAH concentrations were observed in muddy soils because their catabolism by microbes is facilitated in aerobic environments. PAH half-lives are longer at reduced redox potential and because of their reduced porosity; muddy sediments tend to have lower oxygen exchange and therefore reduced redox potential. This is another example of the need for EPA to take a more systematic and rigorous approach in the risk assessment. If there are studies demonstrating higher PAH loss rates from creosote treated wood into muddy sediments, then those studies should be cited and an analysis undertaken to quantify the increases so that the environmental response can be assessed.

EPA's assertion in the forth paragraph of this section that creosote derived PAH have been shown to migrate 150 meters from utility poles should be substantiated with citations. EPRI (1997) found that soil concentrations of PAH decreased from an arithmetic mean of 630 mg ΣPAH/kg dry soil within 0.075 m (8 inches) of treated utility poles to 12.0 mg ΣPAH/kg dry soil at 0.46 m distance and 1.8 mg ΣPAH/kg at 0.76 m distance. Soil concentrations of PAH at 1.5 meters (48") were essentially at background (0.21 µg ΣPAH/kg). Geometric mean values showed an even more pronounced decline with distance suggesting no significant increase in PAH at distances $\geq$ 18" (0.46 meters). EPRI (1997) is not referenced in the EPA's RED.

3.4. Biodegradation. Polycyclic aromatic hydrocarbons form a family of compounds and the routes of degradation and fates are different for the major classes of PAH. In water, PAHs evaporate, disperse into the water column, become incorporated into bottom sediments, concentrate in aquatic biota, or experience chemical and biological degradation. Borthwick and Patrick (1982) estimated the chemical and biological half-life of the dissolved components of marine grade creosote at less than one week in laboratory experiments. More recently, Bestari *et*

al. (1998a, 1998b) observed an exponential decline in creosote derived PAH released into microcosms. The concentration of PAH in these microcosms reached background levels by the end of their 84-day study.

The most important degradative processes for PAHs in aquatic environments are photo-oxidation, chemical oxidation, and biological transformation by bacteria and animals (Neff 1979). Most PAHs in aquatic environments are associated with particulate materials and only about a third are present in dissolved form. Dissolved PAHs will likely degrade rapidly through photo-oxidation (EPA 1980). They degrade most rapidly at higher concentrations, at elevated temperatures and oxygen levels, and at higher levels of solar irradiation. Different PAHs vary significantly in their relative sensitivity to chemical and biological degradation.

Because of their low aqueous solubility and hydrophobic character, the higher molecular weight PAH readily adsorb to particulate materials and solid surfaces in water. The ultimate fate of PAHs that accumulate in sediments is believed to be biotransformation and degradation by bacteria, fungi and algae (EPA 1980; Borthwick and Patrick 1982; Cerniglia 1984; Boldrin *et al.* 1993). Low molecular weight PAHs, such as naphthalene, degrade rapidly, while the higher molecular weight PAHs such as benz(a)anthracene and benzo(a)pyrene are more resistant to microbial attack. Herbes (1981) reported turnover times for naphthalene, anthracene and benz(a)anthracene of 13, 62 and 300 hours respectively. Mueller *et al.* (1991) found that natural microbial communities mineralized 94% of the low molecular weight PAH in 14 days but only 53% of the high molecular weight PAHs was degraded during the same period. They also noted that the most rapid biodegradation of PAHs occurred at the water/sediment interface. This is because prokaryotes oxidize PAH as a first step in metabolism. Deeper sediments usually contain little oxygen, thus inhibiting microbial metabolism.

Saylor and Sherrill (1981) and Cerniglia and Heitkamp (1991) summarized the available literature describing the half-life of PAH in aquatic environments. The results were highly variable and depended on PAH species together with a range of environmental and biological factors such as temperature, the presence of cometabolites, the nature of the microbial community and the availability of oxygen. A broad range of bacteria and fungi have been observed to rapidly degrade numerous light and heavy molecular weight PAH (Grifoll *et al.* 1994; Stringfellow and Aitken 1994; Cerniglia and Heitkamp 1991). Bacterial communities in polluted areas metabolize PAH more quickly than communities in unpolluted areas and lighter weight PAHs are metabolized more quickly than heavier PAH (Herbes and Schwall, 1978). Naphthalene has a short turnover time of hours to days, whereas the five ringed benzo(a)pyrene may have a very long turn over time of years under the most unfavorable conditions. However, Kanaly and Bartha (1999) demonstrated significant biodegradation of B(a)P in the presence of complex hydrocarbon mixtures. Crude oil, distillates of heating oil, jet fuel and diesel fuel supported up to 60% mineralization of 80 µg B(a)P/g soil in 40 days. Millette *et al.* (1995) also demonstrated the interdependence and cometabolism of mixtures of creosote derived PAH following an initial lag time of 5 to 7 days during which the natural microbial community was selected for those phenotypes capable of efficiently metabolizing PAH. In this study, 60 to 75% of the phenanthrene was mineralized within 30 days. This suggested that in the presence of complex cometabolites, phenanthrene, which comprises 19.4% of new creosote oil, may be rapidly lost from the matrix of PAH that move from treated wood into natural environments.

Bogan and Lamar (1995) showed that white rot basidiomycetes are able to degrade a broad spectrum of intermediate (phenanthrene) and heavier creosote derived PAH. Mueller *et al.*

(1989) provide an excellent review of bioremediation technologies designed to remove PAH, including the high molecular weight compounds, from creosote-contaminated sites.

Ingram (1982) observed that the concentration of creosote in leaching vats increased to greater than 700 µg/L in the first 72 hours and then decreased to less than 34 µg/L at the end of 20 days. He attributed that decrease to bacterial metabolism of the low molecular weight PAH being leached from the pile sections in his study. Tagatz *et al.* (1983) noted that creosote concentrations decreased by 42% over an 8 week period in sediments artificially contaminated as part of their mesocosm studies. They attributed the decrease to microbial metabolism.

Neff (1979) attempted to integrate the degradative processes associated with PAH removal from aquatic environments. He concluded that the residence time of PAH in water is brief. The lower molecular weight aromatics (benzene to phenanthrene) are removed primarily by evaporation and microbial activity. Higher molecular weight PAHs are removed mainly by sedimentation and photo-oxidation. Degradation of PAH by animals in the water column is of minor importance. In nutrient rich, biologically active, aerobic, sediments, the degradation of PAH is increased by healthy bacterial and fungal communities. However, in anaerobic sediments, the heavier molecular weight PAH (4 through 7 rings) may persist for years.

The first sentence in this paragraph of EPA's RED is misleading. The literature clearly demonstrates that all PAH degrade under nearly all conditions. The rates are faster in aerobic environments **and in the presence of co-metabolites**; an important subject that EPA completely omitted from its discussion.

In the concluding paragraph under ***Abiotic Degradation***, EPA states that, "However, it should be noted that the photooxidized products of PAHs are stable; therefore, may persist in air/water and soils and become an environmental concern as these photooxidized products are also bioaccumulative." No data or references are presented to substantiate this assertion.

3.5. Bioaccumulation in Fish. Bioconcentration and bioaccumulation of contaminants is of special importance because some aquatic species, most notably bivalves, have demonstrated an ability to rapidly bioconcentrate some contaminants from water. The concern is that persistent contaminants may move up the food chain, biomagnifying to higher concentrations in each trophic level, until contaminants found at non-toxic levels in the ambient environment reach concentrations where they do cause stress and disease. For effective biomagnification and movement of contaminants through the food chain, several conditions must be met. First, organisms must have the ability to bioconcentrate low levels of contaminants from the water column or to bioaccumulate PAH from sediments or their food. Second, these contaminants, or their toxic metabolic intermediates, must be retained, unaltered, in the tissues of the organism until it falls prey to an animal at a higher trophic level.

There are a number of factors that mitigate against biomagnification. If contaminants are not absorbed, they cannot accumulate. Numerous organisms, particularly arthropods and vertebrates, have the ability to rabidly metabolize and/or to excrete organic contaminants. The gut, liver, kidney and gall bladder are common sites of PAH concentration, metabolism and excretion in vertebrates. If the contaminants are rapidly excreted, or metabolized to non toxic compounds, then the chain is broken and biomagnification is not effective in passing contaminants upward through the food chain. DDT is an excellent example of a persistent compound that was bioconcentrated from low levels in the water to higher levels; first in plankton, then in fish, and finally in bird populations with devastating consequences.

3.5.1. Bioconcentration of dissolved PAH. Neff (1982) reported that most aquatic organisms bioconcentrate polycyclic aromatic hydrocarbons (PAH) from low concentrations in the ambient water to higher tissue levels. Bivalve mollusks, particularly the commercially important mussel (*Mytilus edulis*) and oysters of the genera *Ostrea* and *Crassostrea* have received far more attention than other aquatic invertebrates, plants, or fish. They are excellent subjects for monitoring pollutants because they filter substantial quantities of water over large and highly permeable gills. For these reasons mussels have been the subject of numerous studies such as the Global Mussel Watch Program. Many of these studies have focused on the accumulation of metals and the carcinogenic molecule benzo[a]pyrene (B[a]P).

Benzo[a]pyrene levels recorded in Neff (1979) for uncontaminated areas fell in the undetectable to 50 µg ∑PAH/L range. Dunn and Stich (1975, cited in Dunn and Stich, 1976) recorded tissue levels averaging 59 µg ∑PAH/g in mussels from areas associated with marinas and higher levels, averaging 402 µg ∑PAH/g in mussels growing on creosote treated pilings in industrial areas. Dobroski and Epifanio (1980) found that direct uptake of B[a]P from seawater by diatoms was much greater than the rate of trophic transfer from the diatoms to clam larvae.

Polycyclic aromatic hydrocarbon levels in fish are usually low because this group rapidly metabolizes all PAHs (Lawrence and Weber, 1984; West *et al*. 1986a and 1986b) or they excrete them. High concentrations of PAH are typically found in the gut, liver and bile. Raw fish from unpolluted or moderately polluted water seldom contain detectable amounts of PAH. However, smoking and cooking of fish can increase PAH content to significant levels.

Neff (1982) reported bioconcentration factors (BCF) for several PAHs in the clam *Rangia cuneata.* It should be emphasized that the BCF values, which ranged from 6.1 to 32, are for PAH dissolved in water. Eisler (1987) recorded elevated PAH concentrations, especially benzo(a)anthracene, chrysene, fluorene, phenanthrene, and pyrene in oyster tissues and sediments from the vicinity of marinas. Tissue concentrations of PAH were found to be notably higher in cooler months when lipids and glycogen were being stored preparatory to spawning (Marcus and Stokes, 1985). Eisler (1987) summarized BCF values from the literature. The BCF values reported in his own paper contradict his assertion that bivalves accumulate PAHs more rapidly than fish. For all of the values given in his review the average BCFs were:

Bivalves	82 (n = 8)
Fish	6,844 (n=34)

Note: Eisler's (1987) paper reported bioconcentration values from 6 to 236 in the clam *Rangia cuneata.* Four of the five values were less than 33. For fish, bioconcentration values ranged from 44 to 82,916 with most values in the hundred to thousand times' range.

3.5.2. Bioccumulation of PAH from sediments. The ultimate fate of most heavier-molecular weight PAH deposited in aquatic environments is sedimentation. Roesijadi *et al.* (1978) examined the accumulation of Prudhoe Bay crude oil and specific PAH from oil-contaminated sediments by three infaunal invertebrate species, the sipunculid worm *Phascolosoma agassizii* and the clams *Macoma inquinata* and *Protothaca staminea.* They found that the efficiency of PAH uptake from sediments was much lower than from water. Bioaccumulation factors for uptake of the four PAH from contaminated sediments were 0.2 or less, indicating no significant bioconcentration of PAH by this route. However, bioconcentration factors for uptake of these four PAH from seawater were in the 10.3 to 1349 range indicating a

low to moderate potential for bioconcentration from water. Similarly, Driscoll *et al.* (1997a, 1997b) observed steady-state biota-sediment accumulation factors of 0.16 to 0.61 for the freshwater amphipod *Hyalella azteca* and 0.34 to 0.82 for another amphipod (*Diporeia sp.*).

Eisler (1987) suggested that bivalves readily take up PAH from sediments. This hypothesis is contradicted by the results of numerous studies. O'Connor (1991) found that at 117 National Status and Trend Sites where there were both mollusks and fine-grained sediments, the average ratio of mollusk tissue to sediment concentration was only 1.2 for total PAH. He also noted that mollusks accumulate the low molecular weight (and more highly soluble) PAHs to a greater extent (2.0) than the high molecular weight PAH (0.64). Eaton and Zitko (1978) reported that PAH levels in clams and mussels were two orders of magnitude below those detected in sediments and Neff (1979) cited Perdriau's (1964) finding that in no case did benthic animals contain elevated levels of B[a]P when compared with sediment concentrations. Tissue concentrations in the animals were, on average, 36% of the sediment concentrations.

The bioavailability of PAHs is affected by sediment physicochemistry including the proportion of organic carbon, which binds PAH making them unavailable, and the sediment grain size, which affects uptake by sediment ingesting detritivores (Meador *et al.* (1995). Maruya *et al.* (1997) estimated the uptake of sediment contaminants by determining the ratio of contaminant concentration in organism lipid to the concentration in sediment on an organic carbon basis. This work is important in that most modern sediment benchmarks evoked for purposes of protecting biota from excess PAH contamination are based on sediment organic carbon. Maruya *et al.* (1997) observed biota-sediment accumulation factors (BSAFs) varying between 0.0069 and 5.4 confirming the low potential for uptake of PAHs from sediments.

Johnsen (1987) observed that numerous PAH, including anthracene, fluoranthene, pyrene and benzo(a)anthracene, form strong bonds with natural aquatic humic substances. The strength of these bonds increased with time and with the octanol-water partition coefficient. In other words, the higher molecular weight compounds were more tightly bound than the lower weight compounds. White *et al.* (1999) and Tang and Alexander (1999) observed that phenanthrene, anthracene, fluoranthene and pyrene became more tightly bound to sediments and soil humin as time passed. Their hypothesis was confirmed using both mild extractive techniques designed to release only the bioavailable fraction of the PAH and by measured uptake kinetics in plants and animals. Similarly, Haitzer *et al.* (1999), observed decreasing PAH bioconcentration factors in the nematode *Caenorhabditis elegans* in the presence of increased levels of humic substances in soils and sediments. Bioconcentration factors for pyrene decreased from 12,000 in the absence of dissolved organic carbon (DOC) to five to seven thousand at DOC values greater than 10 mg/L. The decreases for benzo(a)pyrene were even more dramatic, decreasing from ca. 35,000 to <5,000 at DOC levels exceeding 15 to 20 mg/L. Similarly, Weinstein and Oris (1999) found that the fluoranthene BCF decreased from 9,054 in the absence of dissolved humic material (DHM) to 2,810 when the water contained 5.0 mg carbon/L. The median lethal time for photoenhanced fluoranthene at a concentration of 4.8 µg/L to fathead minnows (*Pimephales promelas*) increased from ca. 55 hours in the absence of DHM to ca 100 hours in the presence of one or more mg humic acid/L. These authors also reported strong attenuation of UV-A and moderate attenuation of UV-B as a function of increasing humic substances in water.

For mussels, the general trend towards lower levels of higher molecular weight HPAHs relative to the levels in associated sediment supports an uptake mechanism that involves the solution of PAH in water. Supporting this hypothesis is the observed rapid turnover and shorter half-life of the more soluble, lower molecular weight LPAHs in sediments (Dunn, 1980 in Eisler,

1987). This suggests that the more soluble (and more bioavailable) LPAH are effectively removed from sediments and metabolized by bivalves. The higher molecular weight HPAH (associated with chronic stress and genetic disorders) has reduced bioavailability in sediments because of their lower solubility. There is a growing consensus that bioaccumulation of PAH from sediment is attributed to uptake of PAH desorbed from sediment particles into interstitial water. Numerous studies cited above and in Neff (1982) and Meador *et al.* (1995) lead to the general conclusion that sediment-adsorbed PAHs are not readily assimilated by benthic animals. This hypothesis received further support from Swartz *et al.* (1989) who concluded that the concentration of chemicals in interstitial water is the primary determinant of sediment toxicity - not the bulk concentration in the sediment. These historic observations led Swartz *et al.* (1995) to develop a method for assessing sediment PAH toxicity based on predicted interstitial water concentrations of PAH. Both the historical literature and more recent research support the importance of organic carbon in binding aromatic hydrocarbons and reducing their bioavailability.

3.5.3. Depuration of PAH. Southworth *et al.* (1978) found a half-life of less than one hour for all PAHs metabolized by *Daphnia pulex.* Jackim and Lake (1978) reported that the half-life of PAHs in most bivalves is on the order of 2 to 16 days. These studies suggest that PAHs are either rapidly metabolized or excreted - at least by these species.

3.5.4. Biomagnification of PAH in food chains. Neff (1979) reported that the annelid, *Neanthes arenaceodentata,* had little, if any, ability to accumulate 2-methyl-naphthalene from its food. However, the situation is quite different in marine crustaceans and fish where uptake from food was much more efficient than uptake from water. Arthropods (crabs, amphipods, shrimp and etc.) rapidly accumulate the lighter weight PAH and very rapidly excrete or metabolize these compounds. The half life of B[a]P in *Callinectes sapidus* was six days. Neff's (1979) conclusion was that all results dramatically demonstrated the importance of metabolism in eliminating PAH from contaminated crustaceans. Broman, *et al.* (1990) examined the trophic transfer of PAHs in a study involving seston, the blue mussel (*Mytilus edulis*) and the eider duck (*Somateria mollissima*). Contrary to biomagnification, they observed decreasing PAH concentrations with increasing trophic levels. These studies indicate little potential for PAH biomagnification.

3.5.5. Empirical evidence describing the bioaccumulation of creosote derived PAH from treated structures. Goyette and Brooks (1998, 2000) used semi-permeable membrane devices to measure the concentration of 18 dissolved PAH at a distance of 15 cm from individual piling in the Sooke Basin creosote evaluation for Environment Canada. The Σ18 PAH observed in the piling varied between 17.9 and 30.8 nanograms/liter (parts per trillion) in comparison with 13.4 nanograms/liter observed at the reference station. At 14 days post construction, the concentration of 18 priority pollutant PAH in the somatic and lipid rich gonadal tissues of mussels (*Mytilus edulis edulis*) used in *in-situ* bioassays revealed a small increase from 44.12 $\pm$ 8.09 ng ΣPAH in control mussels to 68.07 $\pm$ 9.14 ng ΣPAH in mussels grown within 15 cm of the six piling dolphins. By 185 days post construction, there was no difference in tissue PAH concentrations between mussels grown next to the structures and mussels from the isolated reference area. Interestingly, at the end of the study, tissue concentrations of PAH were actually lower in mussels grown on and next to the creosote treated structure when compared with

controls. At the end of the four year study, the creosote treated structures had developed a diverse and abundant fouling community. Polycyclic aromatic hydrocarbons were not detected above the method detection limit of 20 ng/g in any sample.

Goyette and Brooks (1998) was extensively reviewed by government and academic scientists before being published by Environment Canada. With respect to the use of creosote treated products in aquatic environments, the results of this exhaustive study directly refute EPA's first sentence in the second paragraph of the RED, "In aquatic habitats, fish, shellfish, and crustaceans readily bioaccumulate PAHs from the environment and store these at high levels in the tissues." In Sooke Basin, PAH's were not significantly bioconcentrated by an animal intensively studied by EPA (e.g. *Mussel Watch*) for its ability to bioconcentrate a range of contaminants. While laboratory studies using industrially contaminated sediments and field studies following accidental hydrocarbon spills may result in significant bioaccumulation of PAH, these studies are not applicable to the proper use of a product. The EPA's failure to distinguish between industrial contamination, including accidental hydrocarbon spills, and the use of creosote treated products is a major and consistent flaw in the Creosote RED.

3.5.6. Bioaccumulation summary. Many aquatic organisms are able to efficiently bioconcentrate PAH from the water column. It appears that direct transfer from sediments to organisms living within and on those sediments is minimal. Benthic organisms rarely contain higher concentrations of PAH than are found in the sediments in which they live. PAHs are rapidly metabolized and excreted by vertebrates and arthropods. In bivalves, which do not metabolize PAHs as efficiently as arthropods and vertebrates, the half-life of most PAHs examined was in the range of 2 to 16 days. These data suggest that PAHs are not persistent in the tissues of aquatic species and that movement of PAHs through food chains to higher trophic levels is minimal, if it occurs at all.

Neff (1979) concluded that: "From the limited data available, it would appear that there are large interspecific differences in ability to absorb and assimilate PAH from food. Polychaete worms have a very limited ability to absorb and assimilate PAH, whereas fish absorption of PAH from the gut is limited and variable depending on species of fish, the PAH, and possibly the food matrix in which PAH is administered. Crustaceans, on the other hand, apparently readily assimilate PAH from contaminated food. In all cases where assimilation of ingested PAH was demonstrated, metabolism and excretion of PAH was rapid. Thus, the potential for food chain biomagnification of PAH seems to be limited. For such biomagnification to occur the material must be readily absorbed from food, and once assimilated, it must be relatively resistant to metabolism or excretion." I have seen nothing in recent years that refutes his conclusion.

3.6. Summary for the Red Section, "Creosote – Environmental Fate." This entire section needs to be rewritten with more emphasis on traditional science and intellectual rigor. The literature review is incomplete at best and selective at worst. EPA has not properly focused its attention on the use of creosote treated products. Instead, despite the availability of numerous peer reviewed documents describing the environmental response to creosote, EPA has relied on laboratory studies using whole creosote oil, which is not characteristic of the suite of PAH lost from the product, on information from inappropriate historical industrial practices and hydrocarbon spills – none of which are indicative of the environmental response to the use of properly produced creosote treated wood products. Information specific to the use of creosote treated railway ties and to the environmental response to creosote treated wood used in bridge

construction in freshwater and marine structures has been made available to EPA, but the agency has chosen to ignore this information. Instead, the agency has relied on studies published before year 2000. In closing, this section is woefully missing well supported interpretations of the literature or defensible conclusions. Where conclusions are provided, they are unsupported by relevant citations and in several instances EPA's conclusions are easily rebutted by the literature.

4.0. Environmental Exposure and Modeling. Brooks (2000a), completed a two year study in the Des Plaines River Wetland that dealt first with PAH in wetland sediments adjacent to an operating spur that carried coal into a Commonwealth Edison power plant; and second, a long-term, large scale mesocosm study that quantified the PAH lost from newly treated ties, used ties and a control mesocosm containing untreated oak ties. This study quantified concentrations of the 16 priority pollutant PAH found in limestone ballast; in adjacent wetland sediments; and in stormwater. The study designs and progress were monitored by biologists from the endangered species section of the U.S. Fish and Wildlife Service. The U.S. Forest Service has undertaken peer review, by academics, and the *Railway Tie Study* will be published by the U.S.D.A. Forest Products Laboratory in 2003. A copy of the parent document is appended for EPA's review.

The results of these studies found that PAH migrated from the new railway ties during the summer of the first year following installation when they reached a concentration of 1,052 µg ΣPAH/g dry ballast within 20 cm (7.87 inches) of the tie faces. Nearly all of these PAH remained in the ballast where they were degraded to a mean of 54.56 $\pm$ 91.4 µg ΣPAH/dry ballast by November, 1998. Ballast PAH concentrations declined further to 1.97 µg ΣPAH/g within 5 cm of the tie faces by February 1999. The sum of PAH at all distances (5 cm to 30 cm from the tie faces) remained low adjacent to the weathered ties throughout the study. A second flush of PAH was not observed migrating from the new ties in the second summer of the study and therefore, it appeared that this was a one time event.

One year following installation of the ballast and ties in the mesocosm wetland, small amounts of PAH were found in sediments adjacent to all three rights of way. The highest sediment PAH concentrations observed in each mesocosm were 2.28 µg ΣPAH/g at 5.0 cm from edge of the ballast supporting untreated ties, 3.94 µg/g at 75 cm from the new tie ballast and 2.33 µg/g at a distance of 75 cm from the used tie ballast. Excepting these three values, all other concentrations, including those closest to the new and used tie ballast were $\leq$ 1.8 µg ΣPAH/g dry sediment. All of these sediment concentrations are less than is typically found along any moderately well traveled roadway.

Core sections to a depth of 60 cm indicated that the PAH were retained in the upper 10 cm of the wetland's sediment with little or no additional downward migration. PAH in the highly permeable limestone ballast migrated downward to peak at 60 cm depth followed by a decline at 70 and 80 cm depths. Highest PAH concentrations were observed at 60 cm depth under the untreated ties. However, the ΣPAH at all ballast depths in all treatments was low ($\leq$ 1.291 µg ΣPAH/g)

Benzo(a)anthracene (0.16 to 0.19 µg/L), dibenzo(a,h)anthracene (1.3 µg/L) and phenanthrene (0.58 to 0.66 µg/L) were detected in stormwater once in each of the mesocosms, including the untreated one. Polycyclic aromatic hydrocarbons were not otherwise detected in stormwater on any of the seven stormwater sampling dates. The concentrations of PAH in stormwater were not biologically significant.

The EPA's approach to this assessment, using the GENEEC model reached somewhat similar conclusions. However, the empirical evidence provided above is substantially more convincing.

4.1. Other modeling efforts. Brooks (1996) described a model for assessing the environmental response to CCA-C treated wood. The dilution algorithms for that model were taken from the Creorisk Model, also developed by Brooks (1994). These models depend on worst case analyses and have repeatedly been field tested and found to predict slightly higher environmental concentrations of wood treating preservatives in the water and sediments than have actually been found. The most intensive of these tests occurred in Sooke Basin and EPA is once again invited to read that document. The models provide conservative (with respect to the environment) management tools. They have been used by numerous state and federal agencies to assess the environmental risks associated with the use of treated wood in aquatic environments. To the best of my knowledge, they have never been found to underestimate the water or sediment concentrations of metals or organic compounds lost from treated wood.

5.0. Creosote – Ecological Effects and Environmental Risk Characterization. Contrary to EPA's statement in the Executive Summary, there is an enormous literature available that could be used to assess chronic effects to freshwater invertebrates or to marine/estuarine aquatic organisms. Much of the literature has been developed by EPA scientists. See for instance Swartz (1999). It appears obvious that the Office of Prevention, Pesticides and Toxic Substances has not availed itself of outside expertise – even when that expertise is available within its own agency. Goyette and Brooks (1998 and 2001) and Brooks (2000a) have described in great detail the environmental response to creosote treated wood projects. Additional studies by Bestari *et al.* (1998a, 1998b), Wendt *et al.* (1994) provide a basis for understanding the environmental concentrations of PAH in associated with creosote treated wood and for evaluating the chronic and acute effects these compounds have had on the associated invertebrate communities. This author has not studied the affects of PAH on mammals or birds and my comments are therefore restricted to effects observed in aquatic fish and invertebrates.

5.1. Acute creosote and PAH toxicity in aquatic environments. The diversity of life in aquatic environments attests to species ability to tolerate natural background levels of 1 to 2 µg ΣPAH/L in water and 0.010 to 5.0 µg ΣPAH/g in sediments. The question asked here is, at what level do PAHs cause significant stress and/or pathological responses at the organismal and population levels? Acute toxicity causes observable physiological lesions and is usually measured by mortality. PAH can interact with cells in several ways to cause toxic responses. As an example, they may bind reversibly to lipophilic sites in the cell and thereby interfere with cellular processes. Potentially impacted and important intracellular organelles include lysosomes, which contain strong enzymes important in intracellular digestion of complex organic molecules and in the immune response. Increased lysosomal membrane permeability can result in the unregulated flow of these enzymes into the cytoplasm or blood serum with pathological consequences including autophagy. Eisler (1987) noted that the lower molecular weight, unsubstituted PAH compounds, containing 2 or 3 rings, such as naphthalene, fluorene, phenanthrene and anthracene have significant acute toxicity to some organisms. Whereas the higher molecular weight, four to seven ring aromatics do not. However, these heavier molecules contain potentially carcinogenic and mutagenic intermediates. This is important because the

reader is reminded that much of the low molecular weight PAH are lost during the treating process – significantly reducing the potential acute toxicity associated with creosote treated products when compared with the acute toxicity measured in the raw oil.

5.1.1. Acute toxicity associated with dissolved PAH in marine environments. Goyette and Brooks (1998, 2000) completed an *in-situ* bioassay involving over 2,000 mussels (*Mytilus edulis edulis*) caged at varying distances from six piling dolphins constructed of Class A piling newly treated (over-treated) with creosote to 27 pcf; with eight year old weathered piling; and with untreated piling used as a mechanical control. Additional mussels were caged at a remote reference station in Sooke Basin, British Columbia. Mussel survival was >79 percent in all replicates during the 384 day test and by the end of the study, survival was as high or higher adjacent to the creosote treated structures when compared with the reference station. This study did not document any acute toxicity in marine mussels as a function of distance from the creosote treated structures.

A common measure of acute toxicity is the concentration of a toxicant that causes 50% mortality in a test population within some specified period of time (often 96 hours). This parameter is referred to as the 96-h LC_{50}. Borthwick and Patrick (1982), and Neff (1979) reported 96-h LC_{50} values for several marine animals. These are summarized in Table (1). Interestingly, in Neff's (1979) discussion of the affects of PAH on aquatic animals, he cites Caldwell *et al's.* (1977) finding that continuous exposure to dissolved naphthalene concentrations of 19 to 170 µg/L had no effect on the survival of Dungeness crab larvae. No explanation was given for the lower (8 µg/L) value reported in Neff's (1979) paper or for the differences in the values reported. One might expect that exogenous factors contributed to the differences. The LC_{50} values reported in the literature for most organisms and PAH compounds are in the 500 to 5,000 µg/L range. Neff (1979) found that in all but a few cases, the concentrations of dissolved aromatic hydrocarbons that are acutely toxic to aquatic animals are several orders of magnitude higher than concentrations found even in the most heavily polluted marine and fresh waters. However, sediments from polluted regions may contain aromatic hydrocarbons at concentrations similar to, or higher than those that are acutely toxic. The limited bioavailability of sediment-adsorbed PAHs undoubtedly renders them substantially less acutely toxic than dissolved PAH. He also noted that PAH induced stress is cumulative and exacerbated by exogenous stress factors such as abnormal thermal and osmotic conditions.

5.1.2. Acute PAH toxicity in freshwater. Because PAH heavier than naphthalene are hydrophobic, they are generally found at low concentrations in freshwater and have little potential to create acute or chronic stress in aquatic communities. This statement is not necessarily true for sedimented PAH. Suter and Tsao (1996) and Swartz (1999) summarized conventional benchmarks for priority contaminants in freshwater (Table 2). Freshwater acute fluoranthene toxicity data provided in EPA (1993c) are summarized in Table (3).

Table 1. Acute toxicity of various PAH to marine organisms as measured by 96-h LC_{50} values. All values are in mg/L.

Species	96-h LC_{50}	
mysids (*Mysidopsis bahia*) [1]	18 to 21	
oysters (*Crassostrea virginica*)[1]	700	
pink shrimp (*Penaeus duorarum*)[1]	240	
Sheepshead minnows (*Cyprinodon variegatus*)[1]	3,500	
Mosquito fish (*Gambusia affinis*)[1]	150,000	naphthalene
Mosquito fish (*Gambusia affinis*)[2]	1,180,000	toluene
Dungeness crab larvae (*Cancer magister*)[2]	8	naphthalene
Dungeness crab larvae (*Cancer magister*)[2]	170	naphthalene

Sources: [1] Borthwick and Patrick (1982)
[2] Neff (1979)

Table 2. Summary of consensus LC_{50} values for sediment PAH compounds from Swartz (1999) and lowest daphnid dissolved PAH chronic values reported by Suter and Tsao (1996).

Compound	Swartz (1999) LC_{50} (µg/g)	Suter & Tsao (1996) lowest daphnid chronic value (µg/L)
Naphthalene	71	1,163
Acenaphthylene	15	Not Given
Acenaphthene	23	6,646
Fluorene	90	Not Given
Phenanthrene	155	200
Anthracene	114	<2.1
Fluoranthene	371	15
Pyrene	481	Not Given
Benz(a)anthracene	111	0.65
Chrysene	169	Not Given
Benzo(b)fluoranthene	180	Not Given
Benzo(k)fluoranthene	155	Not Given
Benzo(a)pyrene	179	0.30
Low molecular weight PAH	468	
High molecular weight PAH	1646	
Total PAH	2114	

Table 3. U.S. Environmental Protection Agency (U.S. EPA, 1993c) LC_{50} (mg/L) values describing fluoranthene toxicity to freshwater arthropods.

Species	*Sediment LC_{50} (mg/g organic carbon)*	*Water LC_{50} (mg/L)*
Daphnia magna		3.5
Hyalella azteca	500	44.9
Chironomus tetans	1,587	30.4
Ophiogompus sp.		>178.5
Ophiogompus sp. (UV exposed)		>109.7

Toxicity associated with mixtures of compounds can be additive, antagonistic or synergistic. This is true of mixtures of PAH, which appear to have slightly less than additive toxicity in aquatic organisms. For instance, the LC_{50} for rainbow trout (*Oncorhynchus mykiss*) associated with fluoranthene is >90.5 µg/L. The LC_{50} for rainbow trout subjected to whole creosote oil was nearly ten times higher at 880 µg/L (Polisini, 1994). Pardma *et al.* (1998) examined the toxicity of the water-soluble fraction of creosote to the mysid (*Mysidopsis bahia*) and found median lethal concentrations (expressed as total identified aromatic hydrocarbons) of 180 µg/L. For purposes of evaluating dissolved PAH, revisions of Brooks (1994) have used the ΣPAH model of Swartz *et al.* (1995), which assumes additive toxicity. This discussion suggests that this may be an overly conservative assumption for creosote. However, the author agrees with EPA that more data describing the acute and chronic toxicity to **the suite of PAH actually lost from creosote treated wood products**, would be very useful in better understanding and managing the environmental risks associated with these products.

5.1.3. Photo-enhanced PAH toxicity. The interaction of ultraviolet light (UV) with anthracene and fluoranthene results in modified compounds with increased toxicity to aquatic organisms – at least in laboratory experiments. Landrum *et al.* (1987) reported photoenhanced anthracene LC_{50}'s of 12 µg/L in bluegill sunfish and 1.2 µg/L for *Daphnia pulex.* The authors were unsure as to whether or not the UV light sensitized the target tissues or if it modified the anthracene to a more toxic compound. The observed toxicity was reported to be 400 times greater in the presence of UV than in its absence. Davenport and Spacie (1991) extended these results by demonstrating increased toxicity to *Daphnia magna* associated with a suite of PAH extracted from Lake Michigan sediments. These author's reported that exposure of the sediment elutriates to UV did not result in increased toxicity in subsequent bioassays. Increased toxicity was observed only when the daphnids were cultured in the presence of PAH contaminated elutriate and UV light. Concentrations of PAH in these tests were not reported. Krylov *et al.* (1997) reported on a quantitative structure-activity relationship model (QSAR) predicting the photoenhanced toxicity of 16 PAH. Their model suggested that photoenhanced PAH toxicity is a function of several factoring including the length of exposure to PAH and UV, the relative absorbance of simulated solar radiation (SSR) by each PAH, the resulting quantum yield for formation of triplet-state PAH and the rate of PAH photomodification. They found that toxicity associated with nine PAH compounds was dominated by the PAH modification constant (PMC) and that the photosensitization constant (PSC) was more important in describing toxicity for the remaining seven PAH. This work suggested that photoenhanced PAH toxicity is a function of the particular PAH compounds propensity for modification to a more toxic photoenhanced form and of the target organism's (or tissues') susceptibility to photosensitization. The PSC is likely taxa and life stage specific. This model provides relative toxicity data and not absolute data upon which to determine numerical estimates of toxicity. The authors concluded that photosensitization of target organism tissues and photomodification contribute additively (not synergistically) to photoenhanced PAH toxicity.

Gala and Giesy (1992) reported UV enhanced anthracene toxicity in the green alga (*Selenastrum capricornutum*. The 22-hour EC_{50} for specific growth rate ranged from 37.4 to 3.9 µg anthracene/L depending on the intensity of UV-A radiation. Huang *et al.* (1993) observed similar results for the higher plant *Lemmna gibba* exposed to anthracene, phenanthrene or benzo(a)pyrene in the presence of UV or simulated sunlight. These authors reported the relative toxicity of anthracene to be greater than phenanthrene and both were more toxic that

photomodified benzo(a)pyrene. Growth inhibition was reported at values exceeding thresholds of ca. 200 µg anthracene/L; 500 µg phenanthrene/L and 3,000 µg benzo(a)pyrene/L. The comparatively lower toxicity of phenanthrene (when compared with anthracene) was substantiated by McConkey *et al.* (1997) who hypothesized that the photoenhanced toxicity of phenanthrene is associated with the intermediate product phenanthrenequinone. These authors reported an EC_{50} of 3,500 µg phenanthrene/L in *Lemna gibba* in simulated solar radiation and 10,800 µg phenanthrene/L in visible light (no UV). In contrast, the EC_{50} for the photomodified compound phenanthrenequinone was independent of the presence of UV at 530 to 570 µg/L.

Ankley *et al.* (1995) demonstrated that increased UV enhanced fluoranthene toxicity to *Lumbriculus variegatus* was a function of both dissolved PAH concentration and UV intensity. Oligochaete mortality increased above ca. 29 µg fluoranthene/L in low UV environments. Acute toxicity thresholds were lower under medium light intensity (8 µg/L) and lowest under high intensity UV (75.2 mW/cm2 UV-A) radiation (4 µg/L). These authors noted that *L. variegatus* depurates fluoranthene and that the annelid's physiology includes repair mechanisms that decrease short-term toxicity during periods of darkness. Under medium light intensity (33.5 mW/cm^2 UV-A) mortality did not occur until after 26 hours at a fluoranthene concentration of 60 µg/L. This is important because in the real world, sunlight is intermittent – lasting for only about 16 hours/d at temperate latitudes. Therefore, these values likely overestimate the photoenhanced toxicity of fluoranthene to this species in the real world. Monson *et al.* (1999) observed similar responses in larval frogs (*Rana pipiens*) where increasing mortality was observed in exposures to 3.5 µg/L following exposure to intense light for periods greater than 30 hours. However, photoenhanced fluoranthene toxicity was reported only at much higher levels in the same species by Hatch and Burton (1998). These authors reported an EC_{50} of 276 µg fluoranthene/L in *Rana pipiens,* 247 µg/L in *Ambystoma maculatum* and 52 µg/L in *Xenopus laevis.*

This review indicates that photomodification increases the acute toxicity of several PAH. The increased toxicity appears to be caused by photomodification of PAH and photosensitization of target tissues in an additive manner. Photomodified anthracene appears to be more toxic than that other PAH, including fluoranthene and phenanthrene. In the absence of ameliorative constituents, the threshold for photoenhanced anthracene toxicity has been demonstrated at 1.2 to 4 µg/L. However, humic substances significantly ameliorate photoenhanced PAH toxicity and they absorb UV in the water column.

5.1.4. Acute PAH toxicity to aquatic plants. The affects of various PAHs on aquatic plant growth are highly variable. At low concentrations (10 - 20 µg/L) several PAHs act as a stimulant to plant growth. At 300 µg/L, chrysene was observed by Boney (1974, cited in Neff, 1979) to induce a 58% increase in the growth of the red alga, *Antithamnion plumula.* Other PAHs (anthracene and 2-methylanthracene) caused declines of -20% and -12% in the same alga at 300 µg/L. In general, PAH concentrations greater than 1,000 µg/L inhibit algal growth. As will be seen in this assessment, these PAH concentrations are thousands of times higher than have been observed immediately adjacent to creosote treated structures in open aquatic environments and there is little basis for predicting acute toxicity to aquatic plants associated with these structures.

5.1.5. Summary with respect to acute PAH toxicity in aquatic environments. Acute toxicity associated with dissolved PAH in the water column has not been reported in association with creosote treated structures located in open fresh or marine waters. The review

completed herein indicates that the lowest toxic thresholds are associated with photoenhanced anthracene at 1.2 to 4.0 µg/L. Photoenhanced toxicity has been demonstrated in the laboratory, but not in the field. Observed concentrations of dissolved PAH near creosote treated piling are either not detected or have been detected at <31 ng∑PAH/L. The methodology of Swartz *et al.* (1995) indicated a ∑TU of 0.00074, which is 251 times less than his recommended value of ∑TU < 0.186 for the protection of aquatic life. These observations are consistent with the vibrant invertebrate communities that establish themselves on creosote treated piling (Figure 1) located outside industrial areas where there are numerous sources of many toxic contaminants. Taken altogether this evidence supports Brooks' (1994) conclusion that dissolved PAH associated with creosote treated wood products in open aquatic environments pose no acutely toxic threat to biological resources.

5.2. Chronic toxicity associated with dissolved PAH. Chronic stress can result in reduced growth, reduced life span and/or reduced reproductive output. All of these factors can diminish a population's viability. Neff (1979) addressed chronic stress associated with PAH contamination. He cited Ott *et al.* (1978) and noted that the copepod, *Eurytemora affinis*, suffered statistically significant reductions in the length of life, total number of nauplii produced, and brood size when exposed to 10 µg/L naphthalene, 2-methylnaphthalene, 2,6-dimethylnaphthalene, or 2,3,5-trimethylnaphthalene for the duration of their lives. It should be noted that these naphthalene compounds are the most soluble and therefore the most bioavailable of the suite of PAH found in creosote.

Nearly all PAHs are hydrophobic and lipophilic. Thus, there is a potential for these compounds to associate with stable lipid pools in aquatic organisms. Energy is generally stored as glycogen in bivalves until gametogenesis when the glycogen and lipid stores are converted into eggs and sperm. The eggs contain significant lipid reserves and could become a repository for lipophilic PAH. Moore *et al.* (1989) cited Lowe and Pipe's (1985) observation that long-term exposure to diesel oil at 30 and 130 ppm caused a decrease in the mass of gametes produced by *Mytilus edulis* and *Macoma balthica.*

5.2.1. Reduced Feeding. Mollusks elicit reduced ventilation (feeding) rates at PAH levels as low as 30 to 40 µg/L in seawater (Moore *et al.* 1989). Goyette and Brooks (1998) observed increased tissue concentrations of PAH in mussels grown in an *in-situ* bioassay in Sooke Basin during the first two weeks following construction. Tissue PAH concentrations were not significantly increased above reference levels at any other time during the two year study. Higher concentrations of PAH were found in lipid rich gametes and tissues than in somatic tissues. Subtle effects were seen in the growth of mussels as a function of distance from the piling. The mean increase in mussel valve length was least in mussels grown on the newly treated dolphin (59.3 $\pm$ 2.4 mm); next lowest for mussels grown on the used piling (64.2 $\pm$ 0.9 mm) and highest at the reference station (69.5 $\pm$ 0.8 mm). Mussels caged 2.0 meter downcurrent from the newly treated dolphin grew to a valve length of 67.2 $\pm$ 0.7 mm and those at 10 m distance grew to 68.7 $\pm$ 2.1 mm. The consistent increase in final valve length with increasing distance from the treated wood strongly suggested an influence on the mussel's growth associated with the structures. However, mussel condition factors (dry tissue weight/valve volume) were higher near the creosote treated structures (0.218 $\pm$ 0.015) when compared with the reference (0.177 $\pm$ 0.035). The reduced valve length for mussels in proximity to the creosote treated dolphins may have been caused by a feeding inhibition associated with the narcotic effect

of hydrocarbons, particularly aromatic hydrocarbons. These compounds have a direct effect on cilia, muscles and/or the nervous system, which controls their activity. Reduced feeding rates result in a reduction in "scope for growth," a commonly measured parameter that quantitatively describes the energy available for tissue growth, reproduction and activity. In bivalves, the major problem caused by reduced scope for growth is diminished reproductive capacity. While this does not have immediate consequences at the organismal level, the long-term consequences of reduced recruitment could be significant for the population.

Neff (1979) concluded his discussion of PAH induced chronic toxicity by suggesting that while environmentally realistic dissolved PAH concentrations of 1 - 50 µg/L can cause potentially detrimental, sublethal responses in aquatic organisms, in most cases, the PAH concentrations required to elicit significant sublethal responses are higher than those encountered in all but the most heavily polluted aquatic environments.

Reduced feeding rates were likely the cause of reduced growth in mussel populations as a function of proximity to the Sooke Basin creosote treated dolphins. It should be noted that reduced mussel growth rates were observed in the Sooke Basin study – even when measured water column concentrations of dissolved PAH were in the ng ∑PAH/g range. Goyette and Brooks (1998) hypothesized that creosote derived PAH were primarily transported from the piling to sediments in the form of microparticles of undetermined size. If this hypothesis is substantiated, then it is possible that filtering ob these PAH particles, bringing them into intimate contact with the mussel's ctenidia, caused the reduced mussel growth rather than the uptake of ng/L concentrations of dissolved PAH across cell membranes. This hypothesis has not been investigated.

5.2.2. Reproductive effects associated with creosote treated structures in open aquatic environments. Goyette and Brooks (1998) conducted reproductive bioassays on mussels (*Mytilus edulis edulis*) grown in cages in the immediate vicinity (15 cm) of a major creosote structure in Sooke Basin. Prior to reproductive bioassays, the concentration of PAH in the whole tissues of mussels grown within 15 cm of these structures (21.9 ng ΣPAH/g wet tissue) was not significantly different from that found in mussel tissues from the Open Control (Reference) site (21.7 ng ΣPAH/g). However, approximately twice as much PAH was sequestered in the gonads of ripe mussels at both sites (44.3 ng/g in gonad versus 21.9 ng/g in whole tissue). Reproductive bioassays on all mussel cohorts grown at varying distances from new and aged creosote treated piling and untreated Douglas fir piling did not reveal significant differences. Sixty-five to 89% of all larvae developed normally to the "D" hinge stage. The highest percent normal larval development occurred in the cohort grown immediately adjacent to the weathered piling dolphin. Reproductive effects associated with herring larvae incubated in static laboratory conditions with creosote treated wood reported by Vines *et al.* (2000) are discussed in Brooks (2002).

5.2.3. Summary for dissolved PAH chronic stress associated with creosote treated wood. From the preceding discussion on the uptake of PAH from water, food and sediments, it appears that PAH concentrations in the water column (including interstitial water in sediments) are the parameters of greatest significance in defining chronic stress. The historic literature suggests that sustained water column concentrations of 30 to 50 µg ΣPAH/L can have subtle chronic impacts on populations of marine organisms. These concentrations are approximately 1,000 times greater than those observed in Sooke Basin by Goyette and Brooks

(1998). The work of Goyette and Brooks (1998) indicated reduced mussel growth at much lower dissolved PAH concentrations of <31 g ΣPAH/L. The particulate transport hypothesis of Goyette and Brooks (1998) suggests that uptake of microparticles of PAH passing through the water column in their descent to sediments from creosote treated piling may also contribute to chronic stress. However, it appears that dissolved PAH concentrations do not normally reach those values – except in the case of oil spills or other accidental PAH losses. Goyette and Brooks (1998, 2001) are the only long term studies to quantify chronic effects associated with creosote treated wood structures in open aquatic environments. They observed systematically reduced growth in mussels from 69.5 $\pm$ 0.8 mm at the reference station to 59.3 $\pm$ 2.7 mm in the population of mussels grown with 15 cm of the cluster of pilings. No adverse effects were observed in either survival, which was excellent in all populations or in the two mussel reproductive bioassays completed during the study. In terms of the mussel population, the environmental cost was a reduction in valve length of 10 mm at the end of 384 days in a population that would not have existed absent the habitat provided by the structure.

5.3. Biological response to sedimented PAH. Empirical evidence indicates that much of the PAH lost from creosote treated wood in aquatic environments is deposited in sediments at the base of the structures. As demonstrated in the previous sections, adverse biological responses to dissolved PAH are generally inconsequential because these compounds are hydrophobic and do not readily dissolve in water. This same hydrophobicity causes PAH to bind with dissolved and particulate organic substances – thereby reducing their bioavailability in sediments as well. The biological response to sedimented PAH is well documented.

5.3.1. Acute and chronic toxicity associated with sedimented PAH. It has long been recognized that it is the concentration of PAH in sediment interstitial or pore water that correlates with toxicity – not the bulk sediment concentration of PAH. For instance, Tagatz, *et al.* (1983) found that the lowest creosote concentration at field contaminated sites affecting the abundance or number of mollusks was 844 µg creosote/g dry sediment for mollusks and <177 µg/g for echinoderms, annelids and arthropods. Similarly, Padma *et al.* (1998) reported that the median lethal concentration for *Mysidopsis bahia* in the water soluble fraction (WSF) of creosote extracted from sediments was 700 µg/L compared with a significantly lower level of 180 µg/L obtained when the WSF obtained by direct contamination of water without the mediating influence of sediment.

Pastorok *et al.* (1994) reported sediment ΣPAH concentrations as high as 1,800 µg/g associated with a creosote treating plant at an industrial site in Oregon. They observed significant mortality in *Hyallela azteca* and Microtox™ bioassays within 300 feet of the plant's pier and shoreline. However, significant increases in neoplastic lesions were not observed in the livers of large-scale suckers (*Catastomus macrocheilus*) and no adverse effects on other demersal species were observed outside the highly contaminated nearshore area. Sediments associated with historical industrial activity and spills in Eagle Harbor (Malins *et al.* 1985), the Elizabeth River (Huggett *et al.*, 1992), the Willamette River (Pastorok *et al.* 1994) and Bayou Bonfouca (Catallo and Gambrell, 1987) had been contaminated with greater than 49,000 µg/g of whole creosote oil derived PAH. Acute toxicity and changes in microbial, meiofaunal and macrofaunal communities have been associated with these industrial sites, which have received significant study. The results of some of this research are reviewed in the following paragraphs. It should be emphasized that studies describing the biological response to very high levels of sedimented

PAH associated with historical industrial activity should not be used to infer environmental response to the use of creosote treated wood products.

Acute toxicity has not been associated at sediment concentrations of <10 µgΣPAH/g in open environments (Baekken, 1994; Carman *et al.* 1995, 1997; Wendt *et al.*, 1994; Brooks, 2000a). However, there is evidence of chronic effects, including increased risk of neoplasia, associated with sedimented ΣPAH at concentrations above perhaps 7 to 10 µg ΣPAH/g dry sediment.

Goyette and Brooks (1998, 2001) presented a very large sediment bioassay database collected over a five year period. Their report described the 10-day response of two amphipods (*Rhepoxynius abronius* and *Eohaustorius washingtonianus*) in 69 bioassays completed by Environment Canada in sediments collected at varying distances from the Sooke Basin dolphins. Their report included an evaluation of various methods for assessing effects and an evaluation of various benchmarks available for evaluating the biological response to sedimented PAH (TEL, PEL, ER-L, ER-M, Washington State SQC, Draft EPA SQC, etc.). Additional bioassays conducted included concurrent liquid and solid phase Microtox™ tests and echinoderm fertilization inhibition testing on Day 535 of the study. Sediment concentrations of 18 PAH were determined in each of these bioassays. My point is that EPA has not included any of this data which would be helpful in evaluating the environmental response to sedimented PAH.

5.3.2. Neoplasia associated with polycyclic aromatic hydrocarbons.

Hyperplastic, preneoplastic and neoplastic lesions have been reported in fish for a number of years. These same types of lesions are far less common in bivalves and other invertebrates. Enzymes produced by the cytochromium P-450, Mixed-Function Oxidase (MFO) and Aryl Hydrocarbon Hydroxylase (AHH) systems are responsible for initiating catabolism of lipophilic compounds (including PAH). These systems render hydrophobic molecules more water-soluble and therefore increase their potential for excretion and detoxification. In the case of certain high molecular weight PAH, the intermediate metabolic products are mutagenic and/or carcinogenic. For instance the oxidative catabolism of B[a]P produces arene oxides, some of which bind covalently to DNA and RNA (particularly with guanine). The resulting chromosomal lesions can result in unregulated cell growth and division (cancer).

The ability to metabolize high molecular weight PAH (hPAH) varies significantly between phyla. Among invertebrates, the mollusks have low AHH activity and a limited ability to metabolize hPAH. Arthropods and annelids show increased activity and some marine crustaceans have demonstrated significant cytochromium P-450, MFO and AHH activity.

Vertebrates, including fish, demonstrate high MFO, AHH and cytochromium P-450 capabilities (Varanasi, 1989). The liver is the primary site of MFO activity in fish and the liver, gut and gall bladder are primary sites of PAH concentration, metabolism and excretion. Humans do not normally consume these organs. In Crustaceans, the hepato-pancreas, green gland (excretory organ), pyloric stomach, gills, testes and eyestalks are major sites of PAH accumulation and AHH enzyme activity. Again, these are tissues are not normally consumed by humans, although the hepato-pancreas is sometimes eaten as "crab butter."

Melanomacrophage centers are an integral part of the teleost immune system. Payne and Fancey (1989) observed that the numbers of melanomacrophage centers were increased in the livers of fish exposed to ΣPAH concentrations between 25 to 50 µg ΣPAH/g. Payne, *et al.* (1988) observed changes in mixed-function oxygenase (MFO) enzyme levels and liver fat content in fish exposed to low dissolved hydrocarbon levels of 1000 µg/L (perhaps even as low

as 200 to 300 µg ΣPAH/L). The increased levels of P-450, MFO and AHH enzymes in fish and crustaceans exposed to sediment PAH suggest active catabolism of these molecules. Enzyme induction is not a sign of stress, per se. However there is concern because some of the intermediate products of hPAH catabolism are carcinogenic, mutagenic and teratogenic.

Vogelbein *et al.* (1990) described hepatic neoplasm's' in the Mummichog (*Fundulus heteroclitus*) from a site with 22 µg ΣPAH/g dry sediment. Ninety three percent of the Mummichogs collected at this site had gross hepatic lesions and 33% of these fish had hepatocellular carcinomas. Similar cellular lesions have been described in fish from a highly urbanized area (the Duwamish River estuary) in Puget Sound (Pierce *et al.*, 1977).

Colwell (1986) examined mussels and seawater associated with creosoted marine pilings at the Roosevelt Roads Naval Station Complex in Puerto Rico. She employed *Salmonella typhimurium* in the familiar Ames test (Ames *et al.,* 1975) for mutagenicity and found no detectable mutagenic activity in bacteria from either the water or mollusks associated with the creosote. She concluded that the creosote treated structure did not exhibit any appreciable leaching into the surrounding water.

5.3.3. Bioindicator studies. Are genotoxicity and enzyme induction tests appropriate indicators of environmental risk? There is growing interest in enzyme induction and genotoxicity tests as indicators of environmental risk. However, it is important to understand what these tests actually tell us. Numerous levels of protection mediate affects at the organismal level associated with external factors. Stressful environmental factors such as abnormal temperatures, desiccation, disease organisms, low dissolved oxygen are often avoided by mobile animals such as fish and sessile animals (including many bivalves) isolate themselves within tightly closed valves in an attempt to avoid harmful conditions.

At the next level of protection, an animal's integument isolates internal organs and structures from harmful conditions. The skin and gut epithelia are capable of selective absorption of material. For instance, high molecular weight PAH, adsorbed to sediments, apparently pass through the digestive tract of many annelids without being absorbed through the gut epithelia.

Once foreign materials are absorbed into the blood serum through the skin, gills or gut, organisms respond by sequestering them in vacuoles, metabolizing them in the liver, or cleansing them from the serum as it passes through the kidney. Whether or not a molecule is metabolized or excreted, depends, in great part, on its ability to penetrate cell membranes. The plasma lemma is highly permeable to essential molecules such as glucose, amino acids and lipids. These phospholipid bilayers are not very permeable to ions or to large charged polar molecules. The four to seven-ring hPAH are generally not charged and therefore they pass across the cell membrane and are actively metabolized by vertebrates.

It is well documented that some metabolic intermediates of high molecular weight PAH, particularly arene oxides, can bind covalently to guanine, producing DNA lesions, which may result in unregulated cell growth (cancer). These metabolic intermediates are frequently found in the di gestive gland (liver or hepatopancreas) where metabolism is most active. The literature contains many citations regarding hepatic lesions (including hepatic carcinomas) in demersal fish associated with PAH contaminated sediments. However, the levels of contamination observed at Eagle Harbor, the Duwamish River, Elizabeth River, etc., at which significant increases in hepatic carcinomas have been observed, are generally greater than 25 to 50 µg/g. In some areas, Eagle Harbor sediments contain ΣPAH concentrations as high as 6,000 µg/g.

Mixed Function Oxidases (MFO), Cytochromium P450, Ethoxy Resofurin-O-Deethylase (EROD), and Aryl Hydrocarbon Hydroxylases (AHH) are important enzyme systems for the metabolism of high molecular weight PAH. There are reports in the literature suggesting that PAH metabolizing enzyme systems are activated at sediment PAH levels as low as 1.0 µg ∑PAH/g (e.g. Johnson *et al.* 1994)

As previously stated, intermediate PAH metabolites, such as arene oxides can covalently bind to DNA resulting in lesions. However, DNA contains numerous mechanisms that repair miscoded or damaged sequences. This repair is achieved by a suite of enzymes capable of recognizing damaged or mismatched base pairs and excising them. Environmental and/or random damage to DNA is not unusual and the presence of nicks or double stranded breaks in nuclear (or ribosomal) DNA does not often lead to unregulated cell growth. Increased DNA damage obviously increases the risk for failure of these repair mechanisms resulting in a number of diseases.

The point is that there are numerous levels of protection involved in maintaining the biological integrity of an organism. In evaluating environmental risks, we must recognize the importance of these cellular safeguards. The questions we ask must recognize that different levels of biological organization will respond differently to the same level of insult. Therefore, our questions must be posed carefully and caution should be exercised when extrapolating biological responses at one level of organization to responses at another level. Put simply, the sun feels good on our skin and it is necessary for the synthesis of vitamin D. Peel away the skin and expose the underlying tissue to the same beneficial sun and the underlying cells die. Almost anyone would recognize that evaluating sunlight, based on the response of a naked cell, has nothing to do with an organism's response to the same level of light. This may seem simplistic, but these same principles must be applied to genotoxicity tests.

What question is asked by genotoxicity tests? Ernst (1994) reported the results of genotoxicity tests using subtidal sediments collected at varying distances from a wharf constructed of creosote treated wood. PAH were extracted from the sediments, dried and re-dissolved in dimethylsulfoxide (DMSO). Trout hepatocytes were exposed to varying concentrations of the PAH preparation and genotoxicity assayed using the nick translation assay (NTA) of Gagne and Blaise (1993) and a modified version of the alkaline precipitation assay (APA) described by Olive (1988). The results were quantified by defining a toxicity threshold (TT) as the geometric mean of the Lowest Observed Effect Concentration (LOEC) and the No Observed Effects Concentration (NOEC). This test measured the response of DNA in naked digestive gland cells to isolated PAH suspended in a material, which is an exceptionally powerful solvent for both polar and nonpolar compounds. DMSO is often used as a reaction medium for bimolecular nucleophilic reactions in which the attacking nucleophile (arene oxide) bears a negative charge. Its use in these genotoxicity studies greatly facilitates transfer of PAH across the plasma lemma and of arene oxides into the nucleus.

Based on the preceding paragraphs, it appears that the question being asked is: "How many DNA nicks and breaks occur when we eliminate, or impair, many of a cells nuclear defense mechanisms and expose DNA to PAH and their intermediate metabolic products?" This is an interesting question, and as expected, we find that the degree of DNA insult is proportional to the PAH exposure. In other words, this study revealed a quantifiable dose-response relationship. The dose is isolated PAH and the response is from naked cells whose nuclear and cell membranes have been compromised in the presence of DMSO. Does our current

understanding of bioindicator tests allow their use in assessing environmental risks? There are numerous weaknesses in our current understanding. Consider the following:

Genotoxicity Test Environment	Real World Environment
1. PAH are desorbed and extracted from sediments and dissolved in DMSO.	1. PAH are bound to sediments. They are not readily available in a dissolved form and have reduced bioavailability.
2. No organismal epithelium present	2. After desorption from sediments, PAH must cross ectodermal tissues (skin, gills, gut) before entering the blood stream for delivery to the digestive gland.
3. No kidney present to clear PAH.	3. Kidney present. It functions to clear some xenobiotics. Fish rapidly excrete most PAH.
4. Plasma lemma compromised by DMSO	4. Cell membranes selectively restrict movement of PAH into the cell. This increases the probability of excretion and decreases the probability of metabolism.
5. Lysosomal membranes compromised by DMSO.	5. Lysosomal membranes help contain intermediate metabolites during metabolism.
6. Nuclear membrane compromised by DMSO	6. Nuclear membrane provides another level of protection for DNA.
7. DNA lesions assumed to result in unregulated cell growth.	7. DNA repair mechanisms reduce the probability of unregulated cell growth.

High molecular weight PAH can result in disease in demersal fish at sufficiently high concentrations. The review presented here is intended to provide insight into the mechanisms leading to the observed hepatic carcinomas. However, before these genotoxicity tests can be used to establish environmental criteria, we need to correlate the observed cellular responses with responses at the organismal or population levels of organization. The response of a naked cell, with at least seven layers of protection stripped away, to isolated PAH, does not describe the response of an organ or of whole organisms living in close association with sedimented PAH.

Other bioindicator tests (primarily enzyme induction tests) suffer from the same weakness. The response of an enzyme system to an appropriate substrate has little to do with the response of the organism to that substrate. Bioindicators certainly have a future in environmental studies. However, adequate correlations between cellular or genetic responses and organismal or population responses to pollutant levels have not been made.

Payne, *et al.* (1988) reported a study supporting the hypothesis that many point sources of hydrocarbon contamination could be harmful to fish health. They found that MFO enzyme levels were altered at hydrocarbon levels as low as 1.0 µg ΣPAH/g. The authors noted that PAH levels in this range are encountered over a broad range of aquatic environments, many of which are not associated with pollution. They suggested that hydrocarbons often occur in sufficient concentrations to affect biological responses in fish. Consistent with the discussion presented here, they concluded that meaningful bioindicators must distinguish between effects per se and either chronic or acute effects.

The development of simple, timely, and effective tests to evaluate the risks posed by pollutants to aquatic organisms is important work. However, until a better understanding of the correlation between effects observed in bioindicator studies and the response of organisms and populations living in open environments is achieved, bioindicators have little value as either regulatory tools or for assessing environmental health.

5.3.4. Effects of PAH contamination on populations of aquatic organisms. Mesocosm studies by Stekoll *et al.* (1980), Widdows *et al.* (1982), and Widdows *et al.* (1985) reported similar community responses to petroleum and PAH contamination. Significant, long term reductions in the abundance and diversity of invertebrate fauna were reported when ambient water levels contained 130 µg/L dissolved diesel oil over prolonged periods of time. Less significant population effects were observed on a rocky shore community exposed to 30 µg/L diesel oil for two months.

Tagatz *et al.*, (1983) examined the impacts of creosote-contaminated sand on macrofaunal communities. He found that the lowest creosote (in sediment) concentration, at either of his sites, that affected the number of individuals or species was 844 mg ΣPAH/kg for mollusks and <177 mg ΣPAH/kg for echinoderms, annelids and arthropods.

The adaptation of microbial communities in the gut of *Limnoria tripunctata* and in sediment was discussed in Neff (1979). Similar adaptations were described by Wade *et al.*, (1989) in Gulf of Mexico hydrocarbon seep communities including numerous species of annelids, crustaceans, bivalves and fish. Tissue PAH concentrations indicated that these organisms were chronically exposed to high levels of PAH. The seep organisms were able to survive and thrive in an environment of high PAH exposure. The apparent ability to cope with these elevated levels of PAH may involve specially adapted and/or evolved enzyme systems.

Brooks (2000a) examined the loss of PAH and the biological response to creosote treated bridges crossing Pipe Creek in Indiana and Goyette and Brooks (1998, 2001) reported the results of a long-term environmental risk assessment in Sooke Basin, British Columbia. A review of these studies was provided to EPA in Brooks (2002).

5.4. PAH toxicity summary. The low molecular weight PAHs such as naphthalene and acenaphthene produce acute toxic effects in marine animals because they are more soluble than the higher molecular weight compounds. Acute intoxication in the sensitive larval stages of marine invertebrates may occur at water column concentrations as low as 8 to 10 µg/L. For most species, the literature suggests that water column concentrations of greater that 20 µg/L are required for significant responses. Low molecular weight PAHs are more soluble than the high molecular weight compounds and bacteria and other aquatic organisms more rapidly metabolize them. The potential for their accumulation to toxic levels is small except when introduced in large quantities such as occurs in petroleum spills. Laboratory (including mesocosm) studies

have demonstrated photo-enhanced toxicity associated with dissolved concentrations of anthracene as low as 1.2 to 4.0 µg/L.

Because of their decreased biological availability, sedimented PAHs have a low potential to cause acute pathological responses at either the organismal or population levels in aquatic species. However, sediment levels of creosote <177µg/g have been shown to cause reductions in the abundance of sensitive taxa in field, but not in laboratory studies (Tagatz *et al.*, 1983). Furthermore, bacteria and eukaryotes have demonstrated a remarkable ability to adapt to relatively high levels of background PAH. Chronic toxicity is more difficult to measure than acute toxicity. Chronic stress, resulting in reduced growth, but not in reduced reproductive success in mussels, was reported by Goyette and Brooks (1998) at 31 ng ∑PAH/L concentrations immediately adjacent to creosote treated piling in Sooke Basin. Other reports of chronic stress associated with the use of creosote treated wood products in open environments were not found.

In addition to direct physiological stress, there is a potential for the high molecular weight PAH (particularly B[a]P) to form carcinogenic, mutagenic and teratogenic compounds during metabolism by crustaceans and vertebrates. Neff (1979) summarized his section on neoplasia by noting that while carcinogenic PAH can produce cancer-like growths and cause teratogenesis and mutagenesis in some aquatic invertebrates and vertebrates, there are no reports of the induction of cancer by exposure of aquatic animals to environmentally realistic levels of carcinogenic PAH in the water, food, or sediments. More recent studies describe increases in the number of hepatic lesions and carcinomas with sediment ΣPAH burdens as low as 7 to 10 µg/g.

6.0. Recommended numerical benchmarks for evaluating the environmental risks associated with PAH. Numerous jurisdictions have established benchmarks for evaluating the human health and environmental risks associated with polycyclic aromatic hydrocarbons in aquatic environments. Washington State (WAC 173-204) has published sediment quality standards for individual PAH and for the sum of low and high molecular weight compounds. In addition, the U.S. EPA has proposed, but not adopted, freshwater criteria for acenaphthene, phenanthrene and fluoranthene (EPA 1993a, 1993b and 1993c). This review did not reveal freshwater sediment quality criteria for individual PAH compounds or their mixtures. There are numerous proposals based on the lowest levels at which adverse effects are observed in consolidated databases representing a broad spectrum of environments. The broad application of criteria based on the observance of effects in worst-case environments results in very conservative assessments in those environments not representative of the worst case. The following discussion assumes that the toxicity of mixtures of PAH is additive. As previously discussed, it appears that toxicity associated with the mixture of PAHs called creosote is significantly less than additive – increasing the conservativeness of proposed benchmarks.

6.1. Benchmarks for assessing the risk of dissolved PAH. The ∑PAH model in Swartz et al. (1995) assigns a <5% probability of mortality greater than 24% for all samples with ∑TU <0.186. Swartz *et al.* (1995) stated that, "The ∑PAH Threshold of Acute Toxicity (∑TU = 0.186, $p_{>24}$ = 0.05) is the toxic-unit concentration below which mixtures of PAHs are unlikely to contribute to sediment toxicity and above which PAH mixtures increase $p_{>24}$ over background conditions." Swartz *et al.* (1995) did not distinguish between chronic and acute toxicity in stating that ∑TU = 0.186 is an appropriately protective benchmark. The Swartz *et al.* (1995) model was developed, based on equilibrium partitioning, to estimate sediment toxicity for infauna. More importantly, Swartz *et al.* (1995) compared the ∑PAH model with other PAH benchmarks and found that ∑TU = 0.186 was equivalent to both the Screening Level

Concentration and NOAA's Effects Range-Low (ER-L). Both the SLC and ER-L are sediment benchmarks below which adverse effects (including chronic effects) are rarely observed (lower 10^{th} percentile of the effects database). The SLC and ER-L are frequently used as benchmarks to determine if sediments require further investigation. Contaminant levels at or below the ER-L or SLC are not considered biologically stressful and generally require no further evaluation.

Swartz *et al.* (1995) assumed that additive toxicity for the suite of PAH in creosote. However, there is evidence indicating that the PAH in creosote are less than additive in their cumulative toxicity. The California Environmental Protection Agency (California EPA, 1994) determined 96-hr LC_{50} 's of 990 µg/L for bluegill (*Lepomis macrochirus*) and 880 µg/L for rainbow trout (*Oncorhynchus mykiss*) exposed to whole creosote oil. Assuming that the creosote components were present in the bioassay in proportion to that found in whole creosote oil, these LC_{50}'s represent approximately 112 toxic units. Similarly, Borthwick and Patrick (1982) reported creosote EC_{50} concentrations ranging from 18 µg ΣPAH/L for mysids (*Mysidopsis* bahia) to 710 µg ΣPAH/L for oysters (*Crassostrea virginica*) and 720 µg ΣPAH/L for sheepshead minnows (*Cyprinodon variegates*). Munoz and Tarazona (1993) noted that, "when the sum of individual compounds have to be used, the differences in acute toxicities between individual chemicals (and their cumulative action) could be higher than an order of magnitude."

Application of a 0.1 factor to convert the 96-hr LC_{50} of 880 µg creosote/L for rainbow trout to a chronic value suggests that 11.2 ΣTU would be a defensible benchmark for creosote. However, the 0.186 ΣTU benchmark suggested by Swartz *et al.* (1995) provides a very conservative value for use in evaluating the risks associated with dissolved PAH. The ΣTU approach was adopted by Brooks (1994) for assessing biological risks associated with dissolved PAH near creosote treated structures. Previous cautions regarding the apparently antagonistic toxicity of the suite of PAH released from creosote treated piling may make this assumption overly conservative. A different model recognizing this antagonism must await additional aquatic toxicity data for creosote.

6.2. Sediment quality benchmarks for polycyclic aromatic hydrocarbons in aquatic environments. Waterloo (1999) lists freshwater and estuarine sediment quality benchmarks reviewed in Table (4). Swartz (1999) provided a concise summary of the types of existing guidelines and attempted to consolidate various benchmarks into three tiers for which he claimed consensus support. These levels were, Threshold Effects Concentration (290 µg ΣPAH/g organic carbon), Median Effects Concentration (1,800 µg ΣPAH/g OC) and the Extreme Effects Concentration (10,000 µg ΣPAH/g organic carbon). Swartz (1999) noted that the TEC (290 µg/g OC) is the easiest benchmark to interpret because adverse effects cannot be anticipated at values less than this. He notes that values exceeding the EEC are always associated with obvious adverse effects. Swartz (1999) councils that conclusions regarding the ecological effects of sediment contamination, which likely occur somewhere between the TEC and the MEC, should be based on site specific analysis and weight of evidence derived from the three elements of the sediment quality triad. Swartz (1999) proposed *Consensus Guidelines* that appear to resolve some of the current inconsistencies. He described a ΣPAH toxicity threshold that is consistent with the Effects Range Low (ER-L) of Long *et al.* (1995) and a ΣPAH mixture LC_{50} that is similar to the Effects Range Median (ER-M) described by the same authors. Goyette and Brooks (2001) compared sediment concentrations of PAH with the ΣPAH Toxicity Threshold, the ΣPAH Mixture LC_{50} and the mean of these two values in predicting biological risk

associated with creosote derived PAH. Table (5) summarizes the benchmarks derived from Swartz (1999).

Table 4. Summary of freshwater and estuarine benchmarks for polycyclic aromatic hydrocarbons. Data are from website http://bordeaux.uwaterloo.ca/biol447/waterquality/ /sedaquat3.html. All values are in mg PAH/g.

Value (µg/g)	Type	Jurisdiction	Source
100.00 (ΣPAH)	SLCA Severe Effects Level	British Columbia	BCMOELP (1994)
110.00 (ΣPAH)	SLCA Severe Effects level	Ontario	Persaud *et al.* (1992)
13.30 (ΣPAH)	Recommended Threshold Concentration	United States	Ingersoll *et al.* (1996)
2.00 (ΣPAH)	OMOE SQG – Lowest Effect Level	Ontario	Persaud *et al.* (1992)
22.00 (ΣPAH)	AETA Apparent Effects Threshold	British Columbia	BCMOELP (1994)
4.00 (ΣPAH)	WEA Effects Range Low	British Columbia	BCMOELP (1994)
3.93 (ΣPAH –OC)	∑PAH Threshold Effects Level		Swartz (1999)
21.14 (ΣPAH –OC)	SPAH Mixture LC_{50}		Swartz (1999)
205.00 (ΣPAH)	AET (estuarine)	Mississippi	Lytle & Lytle (1985)
4.00 (ΣPAH)	Effects Range-Low	NOAA	Jones *et al.* (1997)
44.80 (ΣPAH)	Effects Range-Median	NOAA	Jones *et al.* (1997)
13.30 ∑LPAH & HPAH	AET (estuarine and marine)	Washington	WAC 173-204

Table 5. Summary of the å PAH Toxicity Threshold, å PAH Mixture LC_{50} and the mean of these two values for 17 parental PAH. All values are mg-g^{-1} organic carbon. Values, excepting the mean, are from Swartz (1999).

PAH Compound	∑ PAH Toxicity Threshold	∑PAH Mixture LC_{50}	Mean
Naphthalene	13	71	42.0
Acenaphthylene	3	15	9.0
Acenaphthene	4	23	13.5
Fluorene	17	90	48.5
Phenanthrene	29	155	92.0
Anthracene	21	114	67.5
Fluoranthene	69	371	220.0
Pyrene	90	481	285.5
Benz(a)anthracene	21	111	66.0
Chrysene	31	169	100.0
Benzo(b)fluoranthene	33	180	106.5
Benzo(k)fluoranthene	29	155	92.0
Benzo(a)pyrene	33	179	106.0
Low molecular weight PAH	87	468	277.5
High molecular weight PAH	306	1646	976.0
Total PAH	393	2114	1253.5

It should be emphasized that other than the Washington State SQS (WAC 173-204), none of the benchmarks given in Tables (4 or 5) are enforceable sediment quality standards. They are simply guideposts for evaluating the effects of contaminants in sediments. Draft Rule Amendments to the Washington State SQS were distributed in June of 1999. The new standards

include an increase in the low molecular weight PAH standard from 370 mg/kg OC to 593 mg/kg OC and a decrease in the high molecular weight PAH standard from 960 mg/kg OC to 900 mg/kg OC. The sum of these two classes of PAH is proposed to increase from 1330 mg TPAH/kg OC to 1493 mg TPAH/kg OC.

Goyette and Brooks (1998) examined the effects of creosote treated piling in Sooke Basin, British Columbia. The extensive physicochemical and biological database, included infaunal community analysis and *in-situ* and laboratory bioassays using the amphipods *Rhepoxynius abronius* and *Eohaustorius estuarius,* liquid and solid phase Microtox™, echinoderm fertilization and *Mytilus edulis edulis* growth, survival and reproductive tests. Physicochemical analyses included a detailed description of sediment and water column concentrations of alkylated and parental polycyclic aromatic hydrocarbons. This database allowed for an examination of the efficacy of existing and proposed sediment quality benchmarks in predicting adverse biological response. The U.S. EPA draft sediment quality criteria for acenaphthene (130 µg/g organic carbon), phenanthrene 180 µg/g organic carbon) and fluoranthene (620 µg/g organic carbon) were found to be underprotective in that they failed to predict observed adverse biological effects in 3 database samples. False negative responses (adverse effects observed but not predicted by the benchmark) were not observed for any of the other benchmarks. Goyette and Brooks (1998) found 60 instances where individual PAH compounds exceeded the Threshold Effects Level but where no toxicity was observed (TEL described by Jones *et al.*, 1997). These false positive indications associated with the TEL were observed for every PAH compound except naphthalene. The Washington State Sediment Quality Criteria (WAC 173-204) were most efficient in predicting adverse effects (12 false positive responses) and the Probable Effects Level (PEL) resulted in 21 false positive responses. The mean of the TEL and PEL ((TEL + PEL)/2) resulted in 30 false predictions of adverse effects where none were observed. These results suggested that the mean of the TEL and PEL, the PEL or the Washington State AET based SQC were protective and efficient. In contrast, the TEL and the ER-L were not efficient and were considered overprotective in the Sooke Basin environment.

Several recent studies have suggested that these levels may not be sufficiently protective in marine environments. Johnson *et al.* (1994) and Horness *et al.* (1998) are considered together in this review because they both relied on the same data. Both papers also relied heavily on the assertion that observance of a biochemical response to PAH implies physiological impairment. The inappropriateness of this assertion was discussed earlier in this paper. As another example of the problems with this idea, consider that prolonged exposure to the sun is known to increase the risk for skin cancer. On the other hand, humans sweat when exposed to the sun or during exercise. It would be inappropriate to conclude that sweating was a sign of increased cancer risk. Enzyme induction is a sign of physiological response – not necessarily a sign of stress or physiological impairment.

The most significant flaw in Johnson *et al.* (1994) and Horness *et al.* (1998) is that they significantly underestimated sediment PAH exposure – at least in Elliott Bay where they reported 10 mg ∑PAH/kg in contrast to Department of Ecology (WDOE 1995) reports of 111.3 to 593 µg ∑PAH/g. Johnson *et al.* (1994) and Horness *et al.* (1998) reported 6 µg ∑PAH/g) in the Duwamish Waterway and 90 µg ∑PAH/g in Eagle Harbor. In reality, Eagle Harbor sediments contain as much as 6,461 mg ∑PAH/kg (Swartz *et al.*, 1989) and the Puget Sound Environmental Atlas (PSWQA, 1992) indicated sediment ΣPAH levels at numerous locations in the Duwamish Waterway >21 µg ΣPAH/kg. In general, higher contaminant concentrations are

found in shallow nearshore areas associated with Seattle's intensely urbanized upland and with numerous waterfront docks and industrial facilities. Concentrations of ∑PAH decline in the middle and outer reaches of Elliott Bay (PSWQA, 1992) where the authors collected their samples. Sediments in these Puget Sound industrial areas also contain high levels of PCBs and metals. Misitano *et al.* (1994) reported much higher concentrations of both high and low molecular weight PAH in these areas than reported by Johnson *et al.* (1994) and Horness *et al.* (1998).

Juvenile English sole (*Pleuronectes vetulus*), which were the subject of Johnson *et al.* (1994) and Horness *et al.* (1998), are found in shallow water in the intertidal zone where sediment concentrations of all contaminants are highest. As they grow, English sole move into deeper water, but tend to seasonally migrate from deep water in the winter to shallow water in the spring. In British Columbia, English sole are known to make extensive migrations of at least 700 miles (Hart, 1973). The point is that English sole in Elliott Bay and the Duwamish Waterway are exposed to a variety of sediment conditions including ∑PAH concentrations that greatly exceed (by one to two orders of magnitude) those reported in these two reports.

Both papers are based on the assumption that the English sole subjected to histopathological examination were exposed to a single sediment concentration of ∑PAH. That simply is not true and while the study does suggest a correlation between exposure to ∑PAH (and the mix of other contaminants found in these industrial areas) and hepatic lesions, it is not appropriate to attempt to quantify the degree of exposure without significant additional study. Furthermore, while the intermediate metabolites of some high molecular weight PAH can create chromosomal lesions, some metals and other organic compounds found in these contaminated sediments are also associated with cancer. Correlation analysis can never be used as unequivocal evidence of a cause and effect relationship.

To summarize, Johnson *et al.* (1994) and Horness *et al.* (1998) noted some of these problems in their own work. The authors suggested that the results warranted a closer look at the protectiveness of existing sediment quality criteria. Lastly it is important to note that the author's could not demonstrate any adverse effect on the population of English sole in their study. Poston (2001) summarizes other critical reviews of these papers.

Misitano *et al.* (1994) worked in highly contaminated sediments found in Eagle Harbor, Commencement Bay and Elliott Bay associated with depositional environments. The sediments are fine grained and except when disturbed by the thrust of large vessels, they remain undisturbed. These characteristics are commonly associated with most (but not all) contaminated sediments. The bioassay protocol used by Misitano *et al.* (1994) contains two elements that make it difficult to compare the results with real world environments. First, the authors swirled 20 grams of sediment and 800 ml of seawater in one-liter glass beakers to begin the bioassays. This likely resuspended the sedimented PAH particles and PAH adsorbed to clay and/or particulate organic matter – greatly increasing its bioavailability in the water column. The PAH particles and fine grained PIM and POM to which the PAH were likely adsorbed would be the last to settle and would have accumulated on the surface of the well sorted sediment at the end of four hours – again unrealistically increasing the exposure of surf smelt larvae to the contaminants. Second, the authors then placed the beakers under continuous fluorescent light at 3240 lux. The photo-enhanced (ultraviolet spectrum) toxicity of anthracene, phenanthrene, fluoranthene and benzo[a]pyrene is known to occur at thresholds of 3 to 12 µg/L (Gala and Giesy, 1992; Landrum *et al.*, 1987; Ankley, *et al.*, 1995). Photo-enhanced PAH toxicity occurs at an order of magnitude lower PAH concentration than occurs in the dark or at reduced light

levels underwater. Photoenhanced PAH toxicity has been well documented in laboratory bioassays and in microcosm studies – but not well documented in natural aquatic systems.

The results of this study are interesting and demonstrate that the observed effects can be associated with larval exposure to a mixture of contaminants in industrial sediments under the conditions imposed (resuspension of contaminated, fine grained sediments in a small volume of water and photo-activation). However, these laboratory conditions are not characteristic of real aquatic environments where sediments are infrequently disturbed to the degree implicit in swirling them in 800 ml of water. In other words, it is not appropriate to infer that the same response is likely in the real world. The literature suggests that there is a reasonable correlation between concentrations exceeding 700 to 1000 µg ΣPAH/g organic carbon and preneoplastic lesions or neoplasia – but not at concentrations less than this. This bioassay would have been much more realistic had the author's placed the sediments in the beaker and then gently flowed water over them for 12 to 24 hours before beginning their bioassay. The purpose of this review is not to diminish the credibility of these studies. As examples of acceptable science, the author's clearly reported what they did and the results they observed. The purpose of this review is to demonstrate that these laboratory studies do not support a hypothesis that adverse effects occur in wild populations of fish at exposures of 0.5 to 2.0 µg ΣPAH/g dry sediment.

7.0. Risk Characterization. The first paragraph of this section is not supported by the literature. The literature reviewed in this response suggests that there are virtually no acute risks to aquatic organisms associated with the concentrations of dissolved PAH found adjacent to creosote treated wood structures found in open aquatic environments. These hydrophobic compounds are simply found at concentrations too low to cause significant effects. Photoenhanced acute toxicity has been demonstrated in laboratory studies at perhaps 2 to 4 µg/L for some PAH. Those values are hundreds of times greater than the concentrations that have actually been observed. I cannot speak for all marine environments in North America, but in the Pacific Northwest, creosote treated piling are typically encased by luxurious communities of invertebrate organisms – most of whom settled there as larvae. If creosote treated wood posed a "high to very high acute toxicity to marine/estuarine invertebrates" as asserted by EPA, then these communities would not flourish as they do. The fouling community residing on creosote treated piling used as a cover for this response is typically of thousands of similar piling in diverse locations throughout the Pacific Northwest.

EPA has not used the available information regarding chronic toxicity to assess the biotic response to creosote. The reduced shell length, but increased condition index in *Mytilus edulis* reported by Goyette and Brooks (1998) and the detailed invertebrate community analysis with same sample PAH analyses for both parental and alkylated PAH are missing from EPA's deliberations.

This author strongly disagrees with EPA's statement that, "Most of the ecotoxicity data found in the open literature for creosote has limited usefulness in a risk assessment, due to the lack of measured concentrations and other quantifiable parameters." Goyette and Brooks (1998, 2000) and Brooks (2000a) provide detailed benthic community analyses, extensive bioassay data and the quantification of priority pollutant PAH for each sample. In addition, these studies include measurements of total organic carbon, sediment grain size, sediment free sulfides and other physicochemical endpoints important in assessing biological responses. Rather than a lack of information, it is EPA's failure to use the available information that results in a poor risk assessment. Finally, EPA needs to learn to use methods other than risk quotients for completing

these assessments. The agencies limited approach greatly limits their ability to understand the risks associated with pesticides and to intelligently regulate them. There are many knowledgeable scientists in EPA that could help improve this product.

8.0. Summary. This author was disappointed with the lack of rigor and completeness in this draft RED. Properly done, these documents could significantly aid society in learning to manage our resources in a sustainable way. As it stands, the draft RED will only encourage debate to fill in the un-necessary voids in the document. EPA needs to basically start over. The Office of Prevention, Pesticides and Toxic Substances needs to avail itself of the science produced since 1999 and it needs to look within and outside the agency for help in assessing that literature.

One of the problems facing those of us who conduct environmental risk assessments is the lack of definitive benchmarks against which to make judgments. This is particularly perplexing when addressing the risks associated with the use of creosote treated wood in open aquatic environments. The reason is that water quality criteria exist for many compounds, but dissolved PAH are not a problem with creosote products. It is sedimented PAH that must be managed in association with creosote treated wood projects and sediment quality criteria, other than those established by Washington State in WAC 173-204, are lacking. Absent well thought-out sediment quality criteria, it is incumbent for each researcher to establish and defend their individually chosen benchmarks. With its wealth of resources, EPA is the government body that everyone looks to for development of appropriate guidelines. Goyette and Brooks (1998) is one of the few reports to use an extensive sediment bioassay database in an effort to evaluate the protectiveness and efficiency of existing benchmarks, Washington State SQC and draft EPA standards. My point is that it is long past time for the EPA to stand-up and address this complex and difficult task.

A rigorous risk assessment should include a review of typical PAH concentrations encountered everywhere and everyday by Americans. This would include PAH's alongside our highways, PAH's (including carcinogenic species) in meats cooked over charcoal, PAH's associated with forest fires and etc. (Johnston and Harrison, 1984; Khalili *et al.* 1995; Prahl *et al.* 1984; USDA, 1980; Wild and Jones, 1995; Hoffman *et al.* 1984, 1985; Dickhut and Gustafson, 1995; Bradley *et al.* 1994). Other sections that are missing involve an evaluation of management tools such as the *Best Management Practices* developed by the Western Wood Preserver's Institute and the Canadian Institute of Treated Wood (WWPI/CITW, 1996) or *Construction Management Practices* such as those described by Lebow and Tippie (2001). Lastly, Brooks (1999, 2002) has proposed generalized guidelines for the use of creosote treated wood as a management tool.

In closing, it is the author's opinion that this document failed to review the available information and it lacks any reasonable degree of rigor. The inadequacies cannot be fully described in the short time allowed by the agency for this response. The preceding comments have been intended only to expose the inadequacy of the document in general terms and to provide some insight into the depth of available information.

Sincerely,

Dr. Kenneth M. Brooks
Aquatic Environmental Sciences

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Glossary and Acronyms

Gravimetric Units. Many papers and reports use different units to express similar terms. The following table provides a guide to these different terms and units as they appear in this paper.

Contaminant concentrations in tissue, water or sediment

µg/L = ng/ml	Micrograms per liter = nanograms per milliliter	= ppb = parts per billion
mg/L	Milligrams per liter	= ppm = parts per million
g/L	Grams per liter = o/oo	= ppt = parts per thousand
µg/g = mg/kg	Micrograms per gram = milligrams per kilogram	= ppm = parts per million
ng/g =	Nanograms per gram	= ppb = parts per billion

Wood preservative retention

Pcf =	Pounds per cubic foot
Kg/m^3 =	Kilograms per cubic meter

Glossary and acronyms

Abundance	Number of a given taxonomic level of organization.
AET	Apparent Effects Threshold employed in Washington State to define enforceable Sediment Quality Standards. Increasing adverse biological effects are observed above the AET concentration.
Amphipod	Freshwater or marine benthic arthropod commonly referred to as a scud in freshwater.
Anadromous	Migratory life style associated with fish in which adults spawn in freshwater and juveniles migrate to marine environments for development to the adult form.
Anaerobic	Lacking free oxygen - anoxic
Anthropogenic	Derived from human activity.
As	The element arsenic
Avoidance	To deliberately move away from a stimulus
AVS	Acid Volatile Sulfides – a measurement of sulfides in sediment released when a sediment sample is incubated in cold hydrochloric acid (see SEM)
Benchmark	A value assigned or proposed for a compound to indicate its relative hazard to an organism, community or ecosystem.
Bioaccumulation	The process by which an organism incorporates an element or compound into their tissues from water or food.
Bioconcentration	The process by which an organism incorporates a compound or element from all environmental routes (e.g., water, food, respiratory surfaces, etc.) into its tissues.
Biodegradation	The processes by which microbes or organisms breakdown complex compounds.

Biodeposits	Organic matter such as dead animals, metabolic waste, pseudofeces, etc. released from an animal or community of animals.
Biomagnification.	The accumulation of contaminants at increasing concentrations in higher trophic levels in the food chain.
Biosynthesis	The formation of organic compounds by living organisms.
Biotransformation.	The biochemical process by which an organism changes the structure of a compound to a different form with a different toxicity characteristic.
BMP	Best Management Practice – as used herein the term implies specific procedures used during the production of treated wood to improve the products environmental performance.
Bulkhead	A structure installed to interrupt and/or reduce wave action along a shoreline.
Carcinogenesis	The multistage process whereby tissues grow in an unregulated manner.
Catabolism	The breaking down of complex organic molecules by living organisms with the release of energy.
Chironomid	A group of aquatic insects commonly referred to as midges.
Chorion	An embryonic membrane external to and enclosing the amnion. The outer covering of a fish egg.
Cometabolite	An organic substance that facilitates the metabolism of contaminants by providing an additional metabolic substrate.
Creosote	An organic pesticide having high PAH content that is used as a wood preservative.
Cumulative Impacts.	The sum of all impacts from a specific action(s) or event(s) in surrounding areas.
Cytotoxicity	Hazardous or poisonous to cells.
Depuration	Cleansing by elimination.
Diffusion	Process wherein a constituent passively moves out of a material (solid, liquid or gas) into the surrounding medium.
Diversity	The number of different species (or other taxonomic levels of organization) present in a community.
Dolphin	An aquatic structure consisting of multiple piles tied together at the top to form a “Teepee” like structure.
Ectodermal	The outer layers of cells that differentiate an organisms from its Environment (skin).
EEC	Extreme Effects Concentration of Swartz (1999). Adverse biological effects are usually observed at concentrations exceeding the EEC. This benchmark is similar to the ER-M or the AET.
Elutriate	Liquid medium which receives additional material in leaching studies.
Epibenthic fauna	The community of organisms living on the surface of sediments or some other structure.
Epibiota	Organisms on the surface of a substance
ER-L	Effects Range – Low = a contaminant concentration benchmark below which adverse effects are not expected.

ER-M	Effects Range – Moderate = a contaminant concentration benchmark above which adverse effects should be expected in sensitive taxa.
Exogenous	External or foreign. Originating outside a living organism.
Exposure	The process defining the relationship between an organism and a contaminant bounded by concentration, duration, and mode of exposure (i.e. inhalation, ingestion, etc.)
Fines	That portion of the sediment grain size distribution $\leq$ 63 µm in diameter encompassing silts and clays.
Food chain	A series of predator – prey relationships defining the production and consumption of organic matter.
Gametogenesis	The physiological process of producing eggs or sperm (gametes).
Glycogen	Animal starch – a branched-chained polysaccharide.
Guideline	A recommended value that should be followed but that does not carry a burden of enforcement.
Hepatic	Referring to the liver
HPAH	High molecular weight polycyclic aromatic hydrocarbons having $\geq$ 4 ring structures.
Immunotoxicity	The process whereby the function of the immune system is affected By a contaminant.
Integument	A covering or coating structure – such as skin.
In vitro	Within an artificial environment (test tube, culture dish, etc.)
In vivo	Within a living organism
Infaunal	Animals living in the sediments
K_{sp}	Solubility product, an expression describing the solubility of an element or compound.
LC_{xx}	An expression denoting the lethal concentration of a compound of some specified period of exposure (e.g. 96 hrs) for a portion (e.g. 50%) of a population. For instance a 96-hr LC_{50} describes the concentration of a contaminant that will kill 50% of the animals in a 96-hour exposure.
Lipophillic	Compounds that are attracted to organic matrices (fat, adipose tissue) due to the compounds lack of polar radicals.
LPAH	Low molecular weight polycyclic aromatic hydrocarbons having $\leq$ 3 ring structures.
Lysozyme	An enzyme that breaks down cell walls.
MEC	Median Effects Concentration of Swartz (1999)
Mesocosm	An experimental environment created on an intermediate scale of perhaps a part of an acre or in several thousand gallons of water.
Methanogenic	Producing methane. An anaerobic metabolic pathway the derives oxygen from the reduction of nitrate.
Microcosm	An experimental environment created in 5-gallon aquaria or other relatively small containers.
Microlayer	A thin layer that forms on the water's surface where buoyant biological and chemical materials concentrate.
Microtox™	A toxicity testing system based on the inhibition of light output from marine photo-luminescent bacteria.

Mutagenesis	The act of creating a mutation.
Narcosis	An effect of deadening of the nervous system.
Necrosis	Tissue death
Neoplasm	A new spontaneous growth of tissue – sometime cancerous
Oligochaete	A freshwater segmented worm
PAH	Polycyclic aromatic hydrocarbons – a suite of naturally occurring compounds comprised of multiple closed carbon rings.
PEL	Probable Effects Level – a sediment benchmark above which increasing adverse biological effects should be anticipated.
pH	Measurement of the free hydrogen ion content on a logarithmic scale from 1 to 14 with a value of 7.0 being considered neutral.
Photooxidation	The exposure to light that results in an oxygen molecule reacting and binding with an organic compound – photodegradation.
Phototoxicity	The process whereby ultraviolet light causes oxidation of organic compounds to a more toxic state.
Phylogenetic	Pertaining to the evolution of life into discrete groups of levels of organization (i.e. species, family, class, etc.)
Plankton	Small marine or freshwater plants and animals that drift with the surrounding water – includes animals with weak locomotory power.
Plasmalemma	The phospholipid and protein bilayer forming the skin of a cell.
Polychaete	Segmented annelids having hairy parapodia.
Prokaryotes	Ancient life forms whose DNA is not organized in a nucleus – such as bacteria.
Pyrolysis	The breaking down of complex molecules by heat.
Pyrolytic PAH	Polycyclic aromatic hydrocarbons formed during the combustion of organic compounds (forest fires, automobile exhaust, fireplaces, etc.).
Retention	The pounds or kilograms of preservative retained in a cubic foot or cubic meter of treated wood. Pressure treated wood retention standards are specified by the American Wood Preservers Association (AWPA) in their annual book of standards. The retention refers only to the treated zone, which is typically the outer 1 to 2 inches of a piling or timber.
Richness	A measure of a community of organisms that depends on the number of taxa and their abundance.
SLC	Screening Level Concentration. Similar to the threshold effects concentration. A benchmark below which adverse biological effects are not expected.
SPMD	Semipermeable Membrane Devices. Sheets of polyethylene that absorb hydrophobic molecules found at low concentration in water. The polyethylene is then digested leaving the contaminant which can be measured to determine very low concentrations.
Sediment	Inorganic and organic material underlying water bodies.
Standard	A promulgated value used to assess compliance with a law or regulation.
SQS	Sediment Quality Standards for contaminant levels that are enforceable by law.

Teleost	Vertebrate (bony) fishes.
ΣPAH	Sum of PAH. An alternate form of expressing TPAH. It is the sum of the 16 priority pollutant PAH compounds found in water or sediments.
ΣTU	Sum of toxic units. Used to evaluate the toxicity of complex mixtures assuming that the toxicity is additive.
TEC	Threshold effects concentration. A sediment benchmark below which adverse effects are not expected in aquatic communities.
TOC	Total organic carbon – the percent, by weight, of a sediment that is comprised or organic carbon.
TPAH	Total polycyclic aromatic hydrocarbons, by weight in water or sediments – usually expressed in µg/g dry sediment. May be normalized to µg PAH/kg organic carbon.
TVS	Total Volatile Solids – the proportion, by weight, of a dry sediment that is lost during combustion at 550 ^{o}C.
Xenobiotics	Organic compounds originating outside living organisms.